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GENERAL PRACTICE SERIES

THE PROBLEM OF THE BLEEDING STATE

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The problem of the patient who bleeds excessively or alleges he bleeds abnormally, is a familiar one. In some cases the bleeding state may be obvious, since purpura, easy bruising or excessive haemorrhage direct one's attention to it. Sometimes when there is a local lesion it may be difficult to know whether to ascribe the bleeding to local or general causes. A patient, for instance, who presents with epistaxis, may have a local lesion in the nose or a generalized bleeding disease, or both these conditions. Patients with haematuria, haematemesis or melaena may present similar problems.
In general there are 2 main groups:

(*a*) *Bleeding State of recent onset*

This normally implies the onset of a new disease, the bleeding being a symptom. Purpura and bruising are the commonest presenting manifestations though bleeding from skin and mucous membranes may also be present. The underlying disease may be one of many but the following conditions occur with considerable frequency:

Idiopathic thrombocytopenic purpura, toxic-allergic purpura (Henoch-Schönlein group), severe blood diseases like leukaemia and aplastic anaemia and disseminated malignant disease. Careful enquiry should be made into possible toxic factors, e.g. drugs the patient has taken (not forgetting the drugs the doctor himself has prescribed) or possible occupational exposure to noxious substances. A careful history should be taken and physical examination done with especial reference to anaemia (out of proportion to blood lost by haemorrhage), lymph-node, spleen or liver enlargement. All these signs might point to possible blood diseases like leukaemia. Bone tenderness is also a valuable sign for which careful search should be made. Examinations of the peripheral blood and the bone marrow examinations may clinch the diagnosis.

Many tests can be done to attempt to find the cause of the bleeding but the most useful one is an assessment of the platelet numbers. If platelets are plentiful on the blood smear, or if no platelets can be seen on a well stained film of peripheral blood, then a platelet count will add little or nothing to the facts already established. If the patient is thrombocytopenic then the bleeding time is generally prolonged, clot retraction and prothrombin consumption are defective, and the tourniquet test will generally be positive. It is rarely necessary to do these tests in patients who have few or no platelets since they are, in the main, merely a manifestation of a thrombocytopenic state.

Obstetrical patients present a separate problem since there are a number of bleeding conditions associated with pregnancy. Local causes must always be considered but occasionally the excessive bleeding is due to a haemorrhagic state. One variety may be associated with fibrinogen deficiency. The blood appears to be incoagulable and haemorrhage is sometimes very severe. It occurs not only *per vaginam* but also presents as purpura, a haematemesis and melaena. Without drastic measures to combat the fibrinogenopenia (combined with rapid delivery in most cases) bleeding may become uncontrollable.

(*b*) *Bleeding State present since infancy or early childhood*

Haemophilia (and its variant Christmas disease which can only be distinguished from haemophilia by specialized investigations in the laboratory) is probably the commonest and most serious member of this group. The active manifestations of bleeding occur only in males. The diagnosis can generally be made on clinical grounds. In about 65% of cases there is a family history of bleeding affecting males and being transmitted through apparently unaffected females. (Note: The absence of a family history is not uncommon and does not exclude the diagnosis). There is no other familial bleeding diathesis inherited in this way. Bleeding is always severe, the tendency to bleed being lifelong. Bleeding episodes generally follow trauma, though apparently spontaneous haemorrhage, especially from mucous membranes, is not infrequent. Patients bruise easily but purpura is not a feature. Haemarthrosis is almost invariable at some time or other in anything but the mildest grades of haemophilia, and careful examination will generally disclose ankylosis of one or more joints.

Laboratory examination is necessary to clinch the diagnosis. The coagulation time is usually prolonged but a normal coagulation time can occur in the less severe grades of the disease. This simple test needs to be rigidly standardized. It should be noted that the bleeding time and the prothrombin time are normal; so are the platelet count, clot retraction and tourniquet tests. These patients do not generally bleed excessively from venipuncture or from a needle prick such as is done in measuring bleeding time. The prothrombin-consumption test and the thromboplastin-generation tests are abnormal but these tests should be left to specialized laboratories.

Other congenital or hereditary bleeding diseases are rare. Some, e.g. factor 5, factor 7 or prothrombin deficiency, affect the prothrombin complex and are disclosed by a prolonged one-stage prothrombin time; others are more difficult to diagnose. In some the capillaries are defective. In hereditary capillary telangiectasia abnormal vascular dilatations which bleed can be seen on skin and mucous membranes. Although the disease is present at birth, active bleeding may not occur until the late teens or even the early twenties. The telangiectasia are often well seen on the tongue or on the buccal mucosa. Epistaxis is perhaps the commonest symptom and can be very severe. The diagnosis is obvious on sight. In another group the capillary abnormality is shown by a prolonged bleeding time in the presence of a normal platelet count and normal coagulation time.

There remain a fairly large residue of patients who have mild symptoms of bleeding and in whom no laboratory abnormality can be disclosed. Bleeding may for instance continue for up to 48 hours after tooth extraction without obvious cause. Many women bruise without obvious explanation. Occasional patients aver that they bleed excessively after trauma but no laboratory confirmation can be obtained. Possibly these are physiological variants of 'normal'. Perhaps, as our knowledge extends, we may be able to find the cause of these, at present unexplained, phenomena.

TREATMENT

As in all diseases diagnosis must always precede therapy. If the patient has a serious blood disease the treatment is as for that disease. Some thrombocytopenic states can be helped by steroid therapy and/or splenectomy, but treatment along these lines should only be considered once the diagnosis is established beyond reasonable doubt. Vitamin K will only aid a proportion of the patients in whom the 'prothrombin' is low. Vitamin K1 is especially useful when the hypoprothrombinaemia is due to drugs of the dicoumarol or phenindione group. Neither vitamin K nor K1 is of any value if the prothrombin is normal. It can be stated dogmatically that calcium is of no value in any naturally occurring bleeding disease. Vitamin C is only of use in the extremely rare case of scurvy.

In haemophilia, treatment is preventive and involves the avoidance of all possible trauma. Major (or even minor) surgery is extremely hazardous. A pint or two of blood is of very little value in controlling the bleeding which occurs in haemophilia. It is to be hoped that one of these days a really potent antihaemophilic substance will become available but, until it does, practitioners would be well advised to handle this problem with extreme circumspection.

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VAN DIE REDAKSIE

ONLANGSE VORDERING INSAKE OORMATIGE BYSKILDKLIERWERKING

Dit word vandag algemeen aangeneem dat slegs die minderheid van pasiënte met oormatige byskildklierwerking aan die beensiekte wat korrek bekend staan as verspreide osteitis fibrosa ly. Die meeste pasiënte presenteer klinies met niersteine, en kom dus vroeg onder die behandeling van die uroloog. Dit is welbekend dat die stene kalsiumoksalaat en fosfaat bevat, dat hulle ondeurstraalbaar is, dat hulle dikwels groot is en vertakkings het, en dat daar 'n sterk neiging tot newebesmetting is. Slegs 'n klein persentasie van sulke stene staan werklik in verband met oormatige byskildklierwerking, maar dit is baie belangrik om dit in laasgenoemde gevalle uit te ken, omdat snykundige ingreep gewoonlik die enigste hoop aanbied om die pasiënt van die dood weens nierversaking te red. Die volgende prosedure moet gevolg word wanneer 'n kalsium-bevattende niersteen ontdek word. 'n Vroegoggend- (of 24-uurlikse) urinemonster word vir kalsium getoets volgens die eenvoudige metode waarby die Sulkowitch-reagens gebruik word. Indien hierdie toets 'n groot hoeveelheid kalsium aandui, moet 'n serummonster getoets word vir sy gehalte aan kalsium, anorganiese fosfor en alkaliese fosfatase. 'n Verhoogde serum-kalsiumgehalte en verminderde fosfor-inhoud kan beskou word as kenmerkend van oormatige byskildklierwerking—mits die berekening natuurlik volkome betroubaar is. Indien die inhoud aan alkaliese fosfatase vermeerder is, moet die skelet en tande met X-strale ondersoek word om vas te stel of daar tekens is dat die beenresorpsie van osteitis fibrosa plaasvind.

Die moeilikheid onstaan waar die uitslae nie heeltemal duidelik is nie. As die urine-inhoud aan kalsium hoog bly (meer as 200 mgm. per 24 uur op 'n gewone dieet) moet die serum-ontleding, indien dit aan die begin normaal was, verskeie male herhaal word voordat oormatige byskildklierwerking uitgesluit kan word; die diagnose van ‚idiopatiese hiperkalsiurie' word dan gemaak. In die verbygaan kan daarop gelet word dat die ietwat ongemaklike kalsiumarm-dieet tans nie meer as nodig of behulpsaam by uitkenning beskou word nie—'n urine-kalsium van meer as 200 mgm. daagliks op 'n normale dieet moet as verdag bereken word. Die volgende moeilikheid ontstaan by dié gevalle waar die beensiekte nie voorkom nie en die serum-kalsium-gehalte verhoog is (dikwels net by tye) maar waar die serum-fosfor binne normale perke bly. 'n Kombinasie van hierdie aard kan by verskeie ander siektes aangetref word, o.a. sarkoïedose, karsinomatose, miëlomatose, beriellose, en die Cushing-sindroom. Hierdie siektes kan gewoonlik maklik uitgeskakel word—mits hulle darem oorweeg word.

EDITORIAL

RECENT ADVANCES CONCERNING HYPERPARATHYROIDISM

It is now widely appreciated that only the minority of patients with hyperparathyroidism suffer from the bone disorder, which is correctly known as generalised osteitis fibrosa. The great majority present with renal calculi, and consequently fall early into the hands of the urologist. As is well known, the calculi contain calcium oxalate and phosphate, are radio-opaque, frequently large and branched, and very liable to secondary infection. Only a small proportion of such calculi are actually associated with hyperparathyroidism, but the recognition of the latter where it exists is extremely important, since its surgical correction is usually the only chance of saving the patient from eventual death from renal failure. The procedure which should be adopted on discovery of a calcium-containing calculus is as follows. An early morning (or 24 hour) specimen of urine is tested for calcium by the simple method using Sulkowitch's reagent. If this test indicates a large quantity of calcium, a sample of serum is then analysed for calcium, inorganic phosphorus, and alkaline phosphatase content. A raised serum calcium and lowered phosphorus may then be considered diagnostic of hyperparathyroidism, provided, of course, that the estimation is completely reliable. If the alkaline phosphates is raised, the skeleton and the teeth should be X-Rayed for evidence of the bone resorption of osteitis fibrosa.

Difficulty arises where the results are less clearcut. When the urine calcium remains high (over 200 mgm. per 24 hours on a normal diet) the serum examinations if at first normal, must be repeated several times before hyperparathyroidism can be excluded, in which case the diagnosis becomes 'idiopathic hypercalciuria'. It may be noted in passing that the rather awkward 'low calcium diet' is no longer thought to be necessary or helpful in the diagnosis—a urine calcium over 200 mgm. per day on normal diet must be considered suspicious. The next difficulty is in those cases without bone disease, in which the serum calcium is elevated (often only intermittently) but the serum phosphorus remains within normal limits. A combination of this sort may be found in various other conditions, including sarcoidosis, carcinomatosis, myelomatosis, beryllosis and Cushing's syndrome.

Soms is sarkoïedose egter baie moeilik en 'n terapeutiese proef-behandeling met kortisoon (wat die oormaat bloed-kalsium van sarkoïed sal verminder, dog nie dié van oormatige byskildklierwerking nie) sal moontlik 'n nuttige toets wees.

Daar sou geen moeilikheid wees nie indien die serum-fosfor sonder uitsondering van lae konsentrasie was by oormatige byskildklierwerking nie, omdat osteomalakie die enigste ander siekte is waarby hierdie verskynsel voorkom.

Dit kom tog soms voor dat die serum-fosforgehalte binne die normale perke bly, en dit is moontlik selfs verhoog as daar nierversaking was. Laasgenoemde verwikkeling lewer ook sekere moeilikhede op, aangesien die nierversaking self (waarskynlik omdat dit die verlies van liggaamskalsium veroorsaak) sekondêre oormatige byskildklierwerking en beenvernietiging veroorsaak. Dit is dus moontlik, en wel gerapporteer, dat 'n pasiënt terselfdertyd of agtereenvolgend aan beide primêre en sekondêre oormatige byskildklierwerking kan ly.[1] Wanneer nierversaking deur 'n primêre nierkwaal veroorsaak word, is die serum-kalsium nooit vermeerder nie.

Is daar enige ander toetse wat 'n moeilike diagnose kan beklink? Reifenstein en sy medewerkers[2] oorweeg drie nuwe toetse. Die eenvoudigste van die drie is die fosfaat-ontberingtoets. Dit is reeds lankal bekend dat 'n groot fosfor-inname die biochemiese afwykings van oormatige byskildklierwerking grootliks kan uitskakel, en hieruit word dit afgelei dat 'n lae fosfaat-inname hierdie afwykings kan beklemtoon. Hierdie spesiale fosfor-arm-dieet word 3-6 dae lank volgehou (dit is logies om die fosfor-absorpsie verder te verminder deur die gebruik van aluminiumhidroksied), en ná die 3-6 dae is dit heel moontlik dat die serum-fosfor by die hiperparatiroïed-pasiënt na die normale gehalte daal, terwyl daar maar min verandering in die urine-fosfor voorkom. By die normale pasiënt sal dit bevind word dat die serum-fosforgehalte maar min verander het, maar die urine-uitskeiding (van fosfor) behoort sterk te daal.

Die tweede toets is, teoreties altans, fundamenteel. Dit berus op die stelling dat dit een van die grondige aksies van die byskildklierhormoon is om die vermoë van die nierbuisies om fosfor te herabsorbeer te verminder—en dus, mits daar geen ander faktore op die spel kom nie, word die urine-uitskeiding van fosfor vermeerder. Die toets meet dus die werklike kapasiteit van die buisies om fosfor te herabsorbeer. Om dit moontlik te maak, word die serumgehalte aan fosfor verhoog deur aar-inspuiting van bufferfosfaat, en die presiese fosfaat-uitskeiding word dan gemeet. Hierdie berekening word dan in verband gebring met die uitskeiding van kreatinien (of, meer akkuraat, die filtreerspoed van die nierliggaampies word volgens insulien-uitskeiding gemeet). Die fosfaat-uitskeiding is hoër as die normale by oormatige byskildklierwerking (omdat die buisies se heropname onder-normaal is).

Die derde toets is die berekening van kalsium-infusie. By hierdie toets word die serum-kalsium kunsmatig verhoog deur die aar-infusie van kalsiumglukonaat (of 'n soortgelyke saggaraat). Om die een of die ander tans onbekende rede, veroorsaak hierdie metode gewoonlik 'n vermeerdering van ten minste 2 mgm. per 100 ml. in die serum-fosfor by 'n normale mens. By oormatige byskildklierwerking veroorsaak dit 'n baie kleiner styging.

Ons het nog nie baie ondervinding opgedoen insake

These conditions are usually easily excluded, provided they are considered, but sometimes sarcoidosis may be very difficult, and a therapeutic trial of cortisone (which will reduce the hypercalcaemia of sarcoid but not of hyperparathyroidism) may be a useful test.

If the serum phosphorus were always low in hyperparathyroidism it would be easy, because in nothing else except osteomalacia is this phenomenon seen. However, it is not infrequently within the normal range, and may even be elevated if renal failure has occurred. The latter state also brings certain difficulties, since renal failure itself may invoke (probably by causing loss of body calcium) secondary hyperparathyroidism and bone osteoclasis. It is thus possible, and has been recorded, for a patient to suffer both primary and secondary hyperparathyroidism either contemporaneously or consecutively.[1] Where renal failure is caused by a primary kidney condition, the serum calcium is never raised.

Where the diagnosis is difficult, can we look to further tests? Three recent ones are considered by Reifenstein and co-workers.[2] The simplest of these is the phosphate deprivation test. It has long been known that a *high* phosphorus intake may largely abolish the biochemical abnormalities of hyperparathyroidism, so it was conjectured that a low phosphate intake might accentuate them. This special low phosphorus diet is continued for 3-6 days (it would seem logical to decrease further the phosphorus absorption by using aluminium hydroxide), by which time the serum phosphorus in the hyperparathyroid subject may have fallen to low levels, while the urine phosphorus has been little affected. In the normal subject, the serum phosphorus will have changed little, but the urine output should become extremely low.

The second test is, theoretically at least, a fundamental one. One of the basic actions of parathyroid hormone is believed to be that of reducing the capacity of the renal tubules to reabsorb phosphorus and so, other things being equal, to increase the urinary phosphorus. The test therefore measures the actual capacity of the tubules to reabsorb phosphorus. To do this, the serum phosphorus is elevated by intravenous injection of buffered phosphate, and the actual phosphate clearance is measured. This may then be related to creatinine clearance (or, more accurately, to the glomerular filtration rate as measured by inulin clearance); the phosphate clearance is higher than normal in hyperparathyroidism (the tubular reabsorbtion being less than normal).

The third test is that of calcium infusion. Here the serum calcium is artificially raised by the intravenous infusion of calcium gluconate (or similar saccharate). For some presently obscure reason, this manoeuvre usually causes a raising of the serum phosphorus in the normal subject by at least 2 mgm. per 100 ml. In the hyperparathyroid this rise is much less in extent.

Not very much experience has yet accumulated regarding

hierdie toetse nie, sodat ons ons tans maar grootliks moet verlaat op die herhaalde berekening van die serum-kalsium en -fosfor om 'n diagnose te bereik. Om verskeie redes is dit nie wenslik om die nek te ondersoek vir 'n moontlike adenoom van die byskildklier nie, maar in enkele gevalle is selfs dit toelaatbaar, bv. by pasiënte met ontwikkelende nierbeskadiging waar daar nog aan die diagnose getwyfel word selfs ná volledige en noukeurige ondersoek.

Op die gebied van oormatige byskildklierwerking was daar onlangs nog twee belangrike vorderings. 'n Hele paar voorbeelde van 'n familie-voorkoms van oormatige byskildklierwerking is gerapporteer.[3] By hierdie gevalle is die oormatige byskildklierwerking veroorsaak deur *veelvoudige* adenome in die paratiroïed, en daar kan ook adenome voorkom in die harsingslymklier (kleurwerende gewasse), en in die pankreas (gewasse van die eilandselletjies wat 'n oormaat insulien veroorsaak). Daar moet dus by elke geval van oormatige byskildklierwerking gesoek word vir spore van gewasse in hierdie ander endokrien-organe. Indien gewasse in hierdie organe gevind word, kan die geneesheer meer as een byskildkliergewas verwag, en die ander familielede van die pasiënt moet ook ondersoek word.

Ten slotte: Die voorkomssyfer van verskeie spysverteringsteurnisse by oormatige byskildklierwerking is ook bereken.[4] By sommige gevalle is 'n dundermsweer ook teenwoordig. Aan die ander kant kan oormatige byskildklierwerking ook sweer-nabootsende simptome veroorsaak; waarskynlik speel die volgehoue hiperkalsemie hier 'n rol. Die les wat hieruit geleer moet word is dat, by chroniese spysverteringsteurnisse sonder 'n duidelike oorsaak, die urine- en serum-kalsiumgehalte altyd bepaal moet word in geval oormatige byskildklierwerking aanwesig is.

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3. Underdahl, L. O., Woolner, L. B. en Black, B. M. (1953): J. Clin. Endocrinol., **13**, 20.
4. St. Goar, W. T. (1957): Ann. Int. Med., **46**, 102.

these tests, so that our present main standby in diagnosis must still be the repeated estimation of serum calcium and phosphorus. An exploration of the neck 'in case' a parathyroid adenoma is present is not good practice for various reasons, but occasionally even this may be permissible in a patient where the diagnosis remains doubtful after full and careful examination, and in whom renal damage is developing.

Two other advances in knowledge concern hyperparathyroidism. Several instances of a familial incidence of hyperparathyroidism have been found.[3] In these cases the hyperparathyroidism is caused by *multiple* parathyroid adenomas, and, moreover, adenomas may be found also in the pituitary (chromophobe tumours) and in the pancreas (islet cell tumours which produce hyperinsulinism). In any case of hyperparathyroidism, therefore, evidence of tumours of these other endocrine organs should be sought. If found, then more than one parathyroid adenoma must be expected, and the rest of the family should be examined.

Finally the incidence of dyspepsia and other gastrointestinal symptoms in hyperparathyroidism has been evaluated.[4] In some cases a duodenal ulcer is present; on the other hand symptoms like those of ulcer may be caused by the hyperparathyroidism—probably related to the continued hypercalcaemia. The moral of this is that in chronic dyspepsia of obscure origin, the urine and serum calcium should be estimated in case hyperparathyroidism is present.

1. Simpson, S. L. and Wilson, R. R. (1955): Brit. Med. J., **2**, 1283.
2. Chambers, E. L., Gordon, G. S., Goldman, L. and Reifenstein, E. C. (1956): J. Clin. Endocrinol., **16**, 1507.
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THE PICKWICKIAN SYNDROME : ANOTHER HAZARD OF OBESITY

There have recently been several reports in the medical press concerning a syndrome of obesity, somnolence, polycythaemia and excessive appetite, with cyanosis, hypercapnia, and even right-sided heart failure without recognizable heart or lung disease.[1-4] As Burwell and his colleagues[4] point out, this association of features has been well known for a long time; it was particularly well described by Charles Dickens in *Pickwick Papers*. Dickens refers to 'A fat and red-faced boy in a state of somnolency'. He is later addressed as 'Young Dropsy', 'Young Opium-Eater' and 'Young Boa Constrictor'. The extraordinary degree of somnolence which may overcome such individuals even while engaged in conversation or actual muscular activity is illustrated further by Dickens:

> 'A most violent and startling knocking was heard at the door; it was not an ordinary double knock, but a constant and uninterrupted succession of the loudest single raps, as if the knocker were endowed with the perpetual motion, or the person outside had forgotten to leave off . . .
>
> 'Mr. Lowton . . . hurried to the door. . . . The object that presented itself to the eyes of the astonished clerk was a boy—a wonderfully fat boy— . . . standing upright on the mat, with his eyes closed as if in sleep. He had never seen such a fat boy, in or out of a travelling caravan; and this, coupled with the calmness and repose of his appearance, so very different from what was reasonably to have been expected of the inflictor of such knocks, smote him with wonder.
>
> ' "What's the matter?" inquired the clerk.
>
> 'The extraordinary boy replied not a word; but he nodded once, and seemed, to the clerk's imagination, to snore feebly.
>
> ' "Where do you come from?" inquired the clerk.
>
> 'The boy made no sign. He breathed heavily, but in all other respects was motionless.
>
> 'The clerk repeated the question thrice, and receiving no answer, prepared to shut the door, when the boy suddenly opened his eyes, winked several times, sneezed once, and raised his hand as if to repeat the knocking. Finding the door open, he stared about him with astonishment, and at length fixed his eyes on Mr. Lowton's face.
>
> ' "What the devil do you knock in that way for?" inquired the clerk angrily.
>
> ' "Which way?" said the boy, in a slow, sleepy voice.
>
> ' "Why, like forty-hackney-coachmen," replied the clerk.
>
> ' "Because master said I wasn't to leave off knocking till they opened the door, for fear I should go to sleep," said the boy.'

A patient of Burwell *et al.* was in the habit of playing poker once a week. On one occasion he was dealt a hand containing 3 aces and 2 kings, known, we believe, as a 'full house'. Since, however, the recipient of this good fortune then fell asleep, he was unable to obtain any advantage from it. A few days later he entered hospital.

Other clinical features of the Pickwickian syndrome include periodic breathing, twitching, cyanosis, and right ventricular hypertrophy with failure. Clubbed fingers have been seen.

Full investigations of pulmonary mechanics in these cases have ruled out a central lesion involving the respiratory centre as the cause of hypoxia and hypercapnia. They have shown that the increase in actual CO_2 pressure produced by prolonged exposure to high levels of carbon dioxide no longer causes the normal increase in respiration. The respiratory centre, apparently, has a reduced sensitivity, which, however, can be reversed by decreasing the CO_2 pressure to a normal level. The great reduction in vital capacity found in these patients is unassociated with any primary cardiac or pulmonary disease, and pulmonary arterio-venous shunt can also be ruled out, since alveolar ventilation is low rather than high and the arterial CO_2 tension is high rather than low.

It is believed that the extreme obesity leads to a low expiratory reserve volume and to a diminished functional residual capacity. In some way it also leads to shallow respiration and subnormal alveolar ventilation. The chest walls of the patients barely move with deep inspiration, perhaps because the rib cage is immobilized by the heavy fat pads as by a binder or cuirass.

Shallow breathing diminishes alveolar ventilation because each breath has first to fill the dead space before the alveoli are reached. The respiratory rate then increases, but this is entirely unable to compensate for the low alveolar ventilation, and carbon-dioxide retention results, together with reduced oxygenation of arterial haemoglobin. Shallow, periodic breathing may set in, with secondary polycythaemia, cyanosis, somnolence and twitching. The right heart is called upon to perform excessive work, against an increased pulmonary artery pressure and with an insufficient oxygen supply for its own musculature. True cor pulmonale results, with peripheral oedema, venous hypertension, hepatomegaly, right axis deviation, and even incomplete right bundle-branch block on the electrocardiogram. This is comparable to the state of affairs in the right heart strain caused by severe kyphoscoliosis unassociated with emphysema. All this has been shown to disappear after a reduction in weight has been achieved.

This syndrome thus illustrates another of the dangers of obesity. The obesity is caused by overeating, and cannot any longer be looked upon vaguely as a 'hypothalamic' disease because of its association with somnolence. The somnolence in this syndrome can be more physiologically explained by deficient gaseous exchange in the blood and also, perhaps, partly by the soporific effects of too much food.

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THE REGULATION OF THE CEREBRAL CIRCULATION*

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The cerebral circulation is peculiarly dependent upon the systemic blood pressure, since it normally tends to follow passively upon changes in arterial pressure to a greater extent than is the case in most other organs. There is little evidence that either vasoconstrictor or vasodilator drugs in pharmacopoeial doses have any effect upon the cerebral blood vessels in man, which are, however, dilated by all the usual products of metabolism (decreased oxygen, increased carbon dioxide, increased acidity and increased temperature) as well as a number of other agents which have recently come to be associated with cellular activity, and are constricted by some of the reverse changes, especially by increased oxygen and decreased carbon dioxide. It would seem that carbon-dioxide tension is the dominant influence in regulating both the tone of the cerebral blood vessels and the activity of the respiratory centre over the ordinary physiological range. This means that, given the requisite head of pressure, the blood flow through the brain is controlled by its metabolic needs of the moment. How closely the blood flow through the brain is regulated, even in some pathological conditions, is illustrated by what happens in hypertension. Kety *et al.* (1948) have shown that in patients with simple hypertension the cerebral blood flow, the oxygen consumption of the brain, and the arteriovenous oxygen difference are all normal. This is brought about by a concomitant increase in cerebral vascular resistance equal to the elevation in the mean blood pressure. Conversely, Dewar *et al.* (1953) found that hexamethonium given to hypertensives did not lower the cerebral blood flow, but this may not be true of patients with premalignant or malignant hypertension (Crumpton *et al.*, 1955).

The peculiarities of the regulation of the cerebral circulation must be to some extent dictated by the fact that the brain is contained within the skull, which functions for all practical purposes as a rigid box. Since the contents of the cranial cavity must remain virtually constant, this must materially limit the range of both expansion and contraction of the cerebral vessels. It seems likely that the histological structure of the cerebral arteries is related to the regulation

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of the cerebral circulation. They have a prominent internal elastic lamina, in which they resemble the coronary arteries, and few elastic fibrils in the media and adventitia. It has been argued by Wolff (1938) that the effect of the well-developed elastic internal lamina is to damp down the pulsation of the vessels. One may suppose that in the circular muscle fibres of the media, which is so poor in elastic tissue, resides the tonic function of contracting in response to internal stress, for example a rise of blood pressure.

THE ROLE OF THE CIRCLE OF WILLIS

Ever since its discovery in the 17th century the circle of Willis has attracted attention. The fact that the 4 arteries which supply the brain, the 2 internal carotids and the 2 vertebrals, should be thus linked together would seem likely to fulfil some biological function. Macdonald and Potter (1951) have made an experimental study of the cerebral circulation in the rabbit, in which the arrangement of the cerebral arteries is similar to that seen in man. Their observations show that the internal carotid artery and the basilar artery share the blood supply to each cerebral hemisphere in such a way that there is normally no interchange of blood between them. The opposing streams of the two arteries meet in the posterior communicating artery at a 'dead point' at which the pressure of the two is equal. Consequently they do not mix there. Similarly the territories of the two internal carotid arteries meet at a 'dead point' in the middle of the anterior communicating artery. If, however, both internal carotids or both vertebral arteries are occluded, blood passes forwards or backwards respectively from the pair which are still patent. There is then a functioning antero-posterior anastomosis in each posterior communicating artery. Similarly, occlusion of one internal carotid artery leads to its territory being invaded by the basilar supply through the posterior communicating artery and by the opposite internal carotid through the anterior communicating artery. The latter can readily be demonstrated in the course of angiography, for when the opaque medium is injected into one carotid, and the opposite one is compressed, the medium normally crosses the middle line through the anterior communicating artery, but this does not occur if the normal flow through the uninjected carotid is allowed to continue.

The clinician is apt to look upon the circle of Willis in the light of the way in which it reacts to occlusion by disease of one of its contributory arteries, but it can hardly have been evolved to provide a compensation for a pathological state. It seems more probable that, speaking teleologically, its purpose is to guarantee that whatever may be the position of the head in relation to gravity and to the trunk, and however, from one moment to another, this may influence the relative flow through either carotid or vertebral artery, these variations are always compensated for at a point distal to these vessels and within the cranial cavity by the freest possible anastomoses before the brain is reached. Hence, the schema which partitions the circle of Willis symmetrically between the areas of supply of the various vessels with 'dead points' between them is an abstract conception, true only when the head is maintained in a certain position of rest, and perhaps not always even then.

There are also other anastomotic channels distal to the circle of Willis, namely, the anastomoses described by Beevor (1909), Shellshear (1927) and Abbie (1934) at the periphery of the cortical fields of supply of the anterior, middle and posterior cerebral arteries. As Abbie writes: 'There is no such thing as a non-anastomotic artery on the surface of the brain although the anastomoses are not always sufficiently large to compensate for occlusion of some of them. Every vessel joins another and the primitive network whence they have all arisen remains intact. No cerebral artery becomes an end-artery until it has entered the brain substance, but once within the brain no artery appears ever to join another'.

There is yet another anastomosis which is much less important in man than in some other mammals, namely, that between the external and internal carotid artery supplies through the orbit. I shall have more to say about this later.

THE CEREBRAL BLOOD SUPPLY IN HYPERTENSION

As I have already said, in benign hypertension the cerebral blood flow remains the same in spite of the rise of blood pressure, a fact which can be explained only if there is a diminution in the calibre of the cerebral vessels proportional to the rise in blood pressure. As Dewar *et al.* have shown, hexamethonium in such cases does not lower the cerebral blood flow. This must be because the cerebral vascular resistance falls parallel with the blood pressure. This, however, takes time and, if the blood pressure is lowered too rapidly, the fall in the cerebral vascular resistance may not keep pace with it, and cerebral symptoms may occur. Crumpton *et al.* in their careful study of the effect of hexamethonium on the cerebral blood flow showed that 13 patients with malignant and premalignant hypertension had an increase of cerebral vascular resistance of 119%. After hexamethonium the blood pressure fell 39% and the cerebral vascular resistance 29%. The cerebral blood flow fell 16%.

The pathogenesis of malignant hypertension remains obscure in the sense that we do not know why it should occur in some patients with hypertension and not in others with an equally high blood pressure. The experimental work of Byrom (1954) suggests that hypertensive encephalopathy is related to a spasm of the cerebral arterioles. Pathologically there is a necrotizing arteriolitis associated with gross oedema of the brain and multiple foci of ischaemic necrosis 150–300μ in diameter, but local examination of the arterioles may fail to reveal the cause of these lesions. The cerebrospinal fluid commonly shows both a raised pressure and a raised protein content. The raised intracranial pressure may be so great that the patient dies of a cerebellar pressure cone. It seems probable that the essential feature of hypertensive encephalopathy is the reaction of the vessel to a sudden rise of blood pressure. The necrotizing arteriolitis and the oedema of the brain are doubtless changes secondary to the functional disturbance. In the early stages the disorder is reversible, and encephalopathy and acute nephritis will often respond well to reduction of the blood volume by venesection. Hypotensive drugs are also effective, but two points must be remembered. First, in long-standing cases of hypertension the changes of hypertensive encephalopathy may be associated with narrowing of cerebral vessels due to atheroma. The symptoms of the two may co-exist and the possible effects of lowering the blood pressure upon a brain which is already subject to the risk of ischaemia has to be borne in mind. The second point is that even when treatment

has been effective in overcoming the encephalopathy some irreversible changes may have taken place.

CEREBRAL ISCHAEMIA DUE TO ATHEROMA

Owing to the complexity of the cerebral blood supply the effects of atheroma may be complex. The first and most obvious is local narrowing, but the effects of such narrowing depend upon its situation in relation particularly to the collateral circulation. When the collateral circulation is good, a major vessel, such as one internal carotid artery, may be completely occluded without any symptoms resulting. Recent work has shown the importance of the relationship between the carotid and vertebral supplies (Hutchinson and Yates, 1956, 1957). A patient with atheromatous narrowing of both these vessels on one side may remain free from symptoms until the internal carotid becomes completely occluded, when the combined supply becomes inadequate not only for the affected cerebral hemisphere, but also for structures in the posterior fossae so that infarction of the ipsilateral cerebellar hemisphere may also occur.

The commonest symptom of cerebral atheroma is the ischaemic cerebral attack. Such attacks may precede a stroke, or follow partial recovery from one, or occur briefly and intermittently for a long time as the sole manifestation of the impaired circulation. The most probable cause of such attacks is that a vessel has become so narrow that its calibre is normally just adequate and becomes inadequate if the systemic blood pressure falls, for example during sleep. It is possible that arterial spasm may sometimes play a part. It is, I think, less likely that such attacks are usually embolic.

A common early symptom of atheroma of the internal carotid is a transitory attack of amblyopia on the affected side. This must surely be due to a temporary fall of blood pressure in the ophthalmic artery. Why, then, does the eye so rarely become permanently blind when the internal carotid becomes completely occluded? Symonds (1955) has plausibly suggested that in most cases a collateral circulation to the orbit *via* the external carotid and internal maxillary arteries becomes established in time to maintain the blood supply to the eye and so preserve vision even though the normal supply to the ophthalmic artery from the internal carotid eventually becomes cut off.

The symptoms of transitory ischaemia of one cerebral hemisphere resulting from atheroma of the internal carotid artery are too familiar to need more than a brief mention. They include periods of mental confusion with subsequent amnesia, generalized epileptic attacks (though in my experience these are not very common), transitory numbness or tingling or weakness of one or other limb on the opposite side of the body or of a hemiplegic distribution and, when the left hemisphere is involved in a right-handed person, transitory aphasic attacks of various kinds. Attacks of myoclonic twitching of a limb are fairly common after a stroke, and usually involve the lower limb, being particularly prone to occur at night. It is tempting to explain these twitches in terms of fluctuations of the collateral circulation to the leg area of the cortex on the damaged side from the opposite internal carotid artery by the anterior communicating artery.

Headache is sometimes a prominent symptom of atheroma of the internal carotid artery. It may precede more serious disability, such as a stroke. It is common immediately after a stroke and sometimes persists for long periods. Symonds has suggested that it is frequently due to dilatation of collateral vessels, and this seems very probable. If so, at least two sources of the headache can, I think, be distinguished. The commoner type probably arises from dilatation of the vessels of the circle of Willis, but another kind of headache may occur, somewhat resembling migraine and associated with dilatation and increased pulsation of the branches of the external carotid artery.

VERTEBRO-BASILAR ISCHAEMIA

We have only recently learned to recognize in their full variety the symptoms of ischaemia within the distribution of the vertebro-basilar supply. These symptoms are sometimes difficult to distinguish from those of internal carotid atheroma, for which they may sometimes be mistaken, though the reverse is less common. Since the supply to the posterior cerebral arteries normally comes from the basilar artery, vertebro-basilar atheroma may cause ischaemic disturbances of one or both temporal and occipital lobes. The symptoms of these include attacks of confusion and disorientation, impairment of memory, which may amount to severe and persistent memory loss, defects of vision, such as teichopsic attacks reminiscent of migraine, transitory or permanent hemianopia, or even complete cortical blindness. Impairment of the blood supply to the brain-stem and cerebellum may lead to a wide variety of symptoms, such as diplopia, dizziness, unilateral, crossed or bilateral numbness or weakness, dysarthria and ataxia of the limbs (Millikan and Sieckert, 1955). Headache when present is most likely to be occipital.

I have mentioned earlier the importance of atheroma of the vertebral arteries in relation to the blood supply to the brain. Their position in their bony canals in the cervical spine and in relation to the neurocentral joints means that they may be narrowed as the result of pressure from osteophytes in cervical spondylosis. The degree of narrowing may be influenced by the position of the head. Consequently we are now beginning to recognize that transitory cerebral symptoms may be produced in this way by turning the head. Giddiness is the commonest such symptom and it is important to remember this cause of giddiness in elderly people with atheroma and with or without hypertension and to distinguish it from vertigo of aural origin.

ANCILLARY DIAGNOSTIC AIDS

The impairment of the blood supply to the eye resulting from atheroma of the internal carotid artery has recently been employed as a diagnostic test. By using a measured pressure upon the eye it may be shown that the blood pressure in the central retinal artery is lower upon the affected than upon the normal side (Milletti, 1950; Svien and Hollenhorst, 1956). Examination of the cerebrospinal fluid often gives useful information. For two or three weeks after a recent cerebral infarction the fluid is likely to have a raised protein and may be xanthochromic. There is often a moderate excess of cells, in which polymorphonuclears may be in the minority. When there is no reason to suspect infection the presence of a few polymorphonuclear cells in the cerebrospinal fluid is a useful indication of recent brain damage of vascular origin.

In cases of atheroma of the internal carotid artery electro-

encephalographic abnormalities are commonly found over some part of the affected cerebral hemisphere. Webster, Gurdjian and Martin (1955) and Gurdjian, Webster and Martin (1957) discuss the diagnostic value of carotid compression in the neck.

Palpation of the carotids should always be carried out. Sometimes it is easy to be sure that there is a difference between the two sides, but this is a test which is often difficult to interpret. Auscultation occasionally reveals a bruit which may be heard either in the neck or on listening over the eye, and is presumably due to narrowing of the internal carotid.

Cerebral angiography may give valuable information. Some hold that it is best avoided as liable to produce a cerebral vascular accident when used in cases of carotid atheroma, especially perhaps if accompanied by hypertension. Most workers who use it regularly, however, regard such risks as slight, and it would seem justifiable to use carotid angiography when it can provide information which could not be obtained in any other way, and which may be essential in order to decide what treatment to adopt. It has been suggested that surgical exploration of the vessel is less risky and can provide as much information. Only angiography, however, can tell us what is happening in the intracranial course of the vessel and its branches.

Vertebral angiography by the method of direct puncture is certainly more risky than carotid angiography in patients with vascular disease and should therefore be used with caution in such cases.

SOME PROBLEMS OF TREATMENT

In the past our attitude to cerebral infarction, threatened or actual, has been largely fatalistic. Even today the limitations of treatment are all too obvious. Nevertheless, we have a much greater understanding than before of the factors involved, and we can therefore see more clearly what should be the aim of treatment, even if the value of particular methods is often still *sub judice*.

Early diagnosis based upon the recognition of the significance of early symptoms and signs, aided when necessary by special diagnostic methods, is likely, as our methods of treatment improve, to make it increasingly possible to avert, or at any rate postpone, the more serious effects of atheroma of the cerebral vessels.

When once an ischaemic stroke has occurred, whether major or minor, it should be borne in mind that the degree of recovery will often depend upon the extent to which a collateral circulation can be established, particularly to what may be termed marginal areas. This in turn will depend upon a patency of the collateral channels, the height of the systemic blood pressure, and the adequacy of the blood itself in respect of oxygen and nutrients, particularly vitamins. Although bed rest will be essential during the stage of shock, or coma, and in the case of patients too severely disabled to sit up, the aim should always be to get the patient to move his sound limbs and to sit out of bed as soon as possible. This is particularly important with non-hypertensive patients, since immobilizing them may lead to a fall of blood pressure which will militate against the establishment of a good collateral circulation.

There is no evidence that any vasodilator drug dilates the cerebral vessels. Aminophylline is sometimes used for this purpose, but such evidence as there is suggests that it may have a vasoconstrictor action on the vessels of the brain (Kety, 1950). If we bear in mind the relationship between the cerebral circulation and the systemic blood pressure, it is clear that vasodilator drugs which do not dilate the cerebral blood vessels may indirectly impair the cerebral blood flow by lowering the systemic blood pressure.

A similar caution applies to the use of hypotensive drugs. Dewar, Owen and Jenkins (1953) have shown that hexamethonium does not lower the cerebral blood flow. This is because when the blood pressure falls as the result of giving hexamethonium there is a corresponding increase in the calibre of the cerebral vessels. These authors, however, point out that if the mean cerebral flow does not alter there will be a slowing of the linear flow rate and a prolongation of the local circulation time. If, therefore, this is already below normal owing to atheroma of a vessel, the use of such a drug as hexamethonium, although it does not diminish the cerebral blood flow as a whole, may increase the risk of local thrombosis. Before using such drugs in the treatment of hypertension, therefore, it is important to look for evidence of cerebral vascular disease in the history and clinical condition of the patient.

Cervical sympathetic block has had a certain vogue in the treatment of cerebral infarction. If the cerebral vessels in man have no important vasoconstrictor nerve supply it is difficult to see how sympathetic block could directly improve the blood flow through the brain. If, however, in any individuals the collateral circulation through the external carotid is of any magnitude, this might presumably be improved by interrupting the vasoconstrictor fibres to it through the cervical sympathetic. The prognosis of untreated cerebral ischaemia is so extremely variable that it is very difficult to assess the significance of the improvement which it has been claimed may follow cervical sympathetic block.

The value of anticoagulants, and hence the indications for their use, are also debatable. Here, again, we need more statistical evidence than is at present available. Clearly the first diagnostic essential is to be sure that the lesion with which we are dealing is ischaemic and not haemorrhagic. Even so, immediately after an ischaemic stroke has occurred, there is an area of brain in which the capillaries are damaged, and there is undoubtedly a risk that anticoagulants may lead to a haemorrhage into such an infarcted area. Since it is doubtful whether they will in other respects do good it would seem unwise to give anticoagulants within at least 3 weeks of an attack of cerebral infarction. Some would use the cerebrospinal fluid as a guide in such cases and withhold anticoagulants as long as the fluid is xanthochromic. The level of the blood pressure must also presumably be relevant to the risk of haemorrhage. The selection of an upper limit must necessarily be somewhat arbitrary, but most workers would regard a severe degree of hypertension as in itself a contra-indication to the use of anticoagulants.

The most plausible argument for the use of anticoagulants is in patients with cerebral atheroma who suffer from transitory ischaemic attacks, which are rightly regarded as a warning that a more severe occlusion lesion may occur. Several American workers have claimed that the number of ischaemic attacks suffered by patients after being put on anticoagulant therapy is very much less than the frequency with which they occurred before, though there is no adequate explanation of this fact. The clearest indication for the use of anticoagulants, subject always to the precautions I have

already mentioned, is perhaps in those patients whose ischaemic attacks indicate atheroma of the vertebro-basilar system, since the prognosis in such cases is always grave, and there is some evidence that it is considerably better in those treated with anticoagulants (Millikan, Sieckert and Schick, 1955).

Finally, a word or two about arterial surgery. Since the commonest site of narrowing by atheroma of the internal carotid artery is just above the bifurcation of the common carotid, the early recognition of this lesion may render it amenable to surgery and, even when the vessel at that site is completely occluded by a thrombus, it may be patent more distally, and the removal of the obstruction may enable the cerebral blood flow through that vessel to be re-established. Here is a promising method of treatment which will obviously be particularly valuable for patients who happen to have a localized lesion (Robb and Wheeler, 1957).

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CAUSES AND MECHANISMS OF HYPERCALCIURIA

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The estimation of urinary calcium is carried out with increasing frequency in many spheres of medical practice. It is, however, in departments dealing with renal stone that the investigation is most oïten performed and patients with so-called hypercalciuria detected. If the term is to be of any value in clinical work, it should in the first instance be clearly understood what is meant by hypercalciuria.

The term hypercalciuria implies an amount of calcium excreted daily in the urine above the values normally found. An upper limit of normal has therefore to be established and amounts in excess of this are regarded as constituting hypercalciuria. In routine work it is not practical to determine the 24-hour urinary output of calcium under standard conditions of diet, and the normal excretion is therefore defined as the amount excreted in 24 hours on an ordinary dietary regime. The normal, as in many biochemical estimations, will be a range of values, and the upper limit of normality under ordinary conditions of diet is the figure to be decided on.

Samson Wright[1] states that the amount of calcium excreted in the 24-hour urine varies from 50 to 250 mg. Cottet and Vittu[2] regarded the upper limit of normal as 200 mg. Ca in the 24-hour urine. Their upper limit of normality would appear to be too low, since 31% of their patients excreted amounts above this figure and were regarded as hypercalciurics. In a series of 162 normal patients investigated by Pyrah and Raper[3] under ordinary conditions of diet, the urinary calcium varied considerably; in 90·8% the 24-hour figure was below 250 mg.

It thus seems reasonable to conclude that the upper limit of normal urinary excretion of calcium is in the vicinity of 250 mg. per 24 hours, and that hypercalciuria may be arbitrarily defined as the excretion of more than 250 mg. of calcium in the 24-hour urine, under ordinary conditions of diet and in more than one 24-hour period. A finding of hypercalciuria, as thus defined, should stimulate further investigation of the patient. The causes are many and varied.

CAUSES OF HYPERCALCIURIA

The causes have been classified in the following manner:

EXTRA-RENAL

Metabolic
- Excessive ingestion of calcium
- Excessive ingestion of vitamin D
- Acidosis
- Recumbency
- Osteoporosis

Hormonal
- Cushing's disease
- Hyperparathyroidism

Systemic
- Sarcoidosis
- Multiple myelomatosis
- Paget's disease
- Metastatic osseous malignancy

RENAL

Secondary
- Infection
- Fanconi syndrome
- Hyperchloraemic acidosis

Primary
- Idiopathic hypercalciuria

Excessive Ingestion of Calcium

This implies a diet excessively rich in calcium-containing foods such as milk and cheese, or an abnormal addition

to the diet of excessive amounts of calcium, e.g. calcium-containing alkali powders. In such cases a reduction of the calcium-containing items in the diet will correct the hypercalciuria.

Excessive Ingestion of Vitamin D

The mechanism here is thought to be an increase in the absorption of calcium from the intestinal canal as a result of the excessive intake of vitamin D. This may occur in the treatment with large amounts of vitamin D of such conditions as resistant rickets, osteomalacia, arthritis, lupus vulgaris and hypoparathyroidism.

Acidosis

A common instance of this is the hypercalciuria which occurs during the administration of large amounts of ammonium chloride. The calcium is excreted in the urine as base to assist in the excretion of the excess of acid.

Recumbency

There is a marked increase in calcium excretion during periods of immobilization.[4] This increase commences a few days after recumbency begins and continues over a long period. It is thought to be due to a decrease in bone formation while bone resorption continues unabated. Hypercalciuria occurs more readily in the presence of sudden causes of cessation of bone formation such as immobilization for a fracture or poliomyelitis with loss of muscle action.[5] The degree of hypercalciuria depends on the discrepancy between bone destruction and bone formation.

Osteoporosis

This may be either senile or post-menopausal in type. Hypercalciuria may be present in the early stages of this condition but will no longer occur after the skeleton is demineralized.

Cushing's Disease

In this condition there is over-production of adrenal corticoid hormone causing osteoporosis and consequent hypercalciuria.

Hyperparathyroidism

This condition is an important cause of hypercalciuria.

There are two main theories on the mode of action of the excess of parathyroid hormone. Thomson and Collip[6] believe that the hormone stimulates osteoclastic activity, leading to widespread lacunar absorption, the liberated calcium and phosphate being absorbed into the circulation and the excess excreted by the kidneys. Jahan and Pitts[7] carried out animal experiments to test this theory and state that the hypercalcaemia (and resultant hypercalciuria), produced by the administration of parathormone are dependant on its extrarenal action of mobilizing calcium from the body stores, and not on any specific depression of renal tubular reabsorption of either calcium or phosphorus. Milne,[8] after experimental observations on human calcium metabolism, supports this theory, and further experimental work has been done by Stewart and Bowen[9] and Talmage *et al.*[10] to substantiate it.

The second theory is that of Albright,[5] who states that the excess of parathyroid hormone affects the renal threshold of the serum phosphates so that an excess is excreted in the urine. The fall in serum phosphate ions results in mobilization of calcium from the bones in order to maintain the solubility product at a constant level. The serum calcium is thus raised and any excess is excreted in the urine.

In both theories the mode of production of the hypercalciuria is explained in the same manner viz. by the excretion of the excess of serum calcium in the urine.

Sarcoidosis

Hypercalciuria is an occasional finding in sarcoidosis. In this condition there is no generalized bone decalcification, and the bone lesions, when present, are in the nature of circumscribed lesions of coarse trabeculation and sharply punched-out small cyst-like areas, chiefly in the hands and feet. Harrell and Fisher[11] found an elevated serum calcium in sarcoidosis, which was not confined to cases with bone disease, in 6 out of 11 cases investigated by them. Albright[5] reported a case with hypercalciuria, and Henneman, Carroll and Dempsey[12] investigated 2 such cases and state that the hypercalciuria is due to the endogenous production of a substance resembling vitamin D. Studies in their cases of sarcoidosis with hypercalciuria showed low calcium output in the faeces. This state could be reversed by giving cortisone. This effect of cortisone has been demonstrated by Dent[13] in cases of idiopathic hypercalcaemia of infancy, sarcoidosis, and ordinary vitamin D intoxication, and it forms the basis of his cortisone test. In this test 150 mg. of cortisone are given for 10 days and serum-calcium levels are estimated on the 5th, 8th and 10th days. The hypercalcaemia of sarcoidosis will rapidly fall during this time, usually to normal levels.

Multiple Myelomatosis

The serum calcium may be raised in multiple myelomatosis and then the amount of calcium in the urine is also high. These findings were present in a case of multiple myelomatosis described by Caylor and Nichol[14] and in a case of solitary myeloma reported by Nassim and Crawford.[15] These findings may give rise to difficulty in the differential diagnosis between multiple myelomatosis and hyperparathyroidism. The serum phosphorus is usually normal or raised in multiple myelomatosis but Snapper[16] reported a case with low serum phosphorus. Bence-Jones protein has been found in the urine in less than half of the cases of myeloma and when it occurs it may be continuous or periodic, and in scanty or large amounts. Bence-Jones proteinuria may also occur in myxoedema, leukaemia and carcinoma. The serum alkaline phosphatase however is usually normal in multiple myelomatosis and a rise, if present, is insignificant compared with that found in advanced hyperparathyroidism.[17] The mechanism of the hypercalcaemia is still unknown. It may be absent in the presence of widespread bone lesions and osteoporosis,[18] or it may be present in the absence of demonstrable bone lesions.[19]

Paget's Disease

Hypercalciuria may occur in this condition when the disease is advancing or when a patient with the disease is immobilized. In the active stage of Paget's bone absorption occurs with somewhat haphazard new bone formation, and calcification of the new bone is incomplete. There is thus a discrepancy between bone destruction and bone formation. As a rule there is no difficulty in establishing the diagnosis.

Metastatic Osseous Malignancy

Malignant secondary invasion of bone may give rise to hypercalcaemia and hypercalciuria. A primary source should be sought for in the common sites, viz. breast, prostate, kidneys, bronchus and thyroid.

Renal Infection

Acute pyelonephritis has been stated by Albright[5] to be an occasional cause of hypercalciuria. The mechanism by which this might occur is tubular damage and resultant faulty tubular reabsorption of calcium, leading to excessive excretion in the urine. This view is supported by a case of hypoparathyroidism described by Albright in which there was no calcium in the urine, but when a severe staphylococcal renal infection supervened the patient started excreting large amounts of calcium and as the condition responded to treatment with antibiotics the urinary calcium gradually returned to zero.

Renal Hyperchloraemic Acidosis

In this condition there is primarily a tubular dysfunction with no glomerular insufficiency. The nature of the primary pathological renal lesion in these cases varies, being either infective or degenerative.

The metabolic breakdown of the foodstuffs of a normal diet leads to a slight excess of fixed anions. These patients are unable to keep in proper acid-base balance and develop a metabolic acidosis with low plasma bicarbonate and high plasma chloride. The tubular defect may be considered to be a partial failure of sodium-hydrogen exchange leading to decreased excretion of ammonia and titratable acid and increased excretion of bicarbonate. Since sodium-hydrogen exchange is a mechanism of sodium reabsorption, one might expect that these patients would become depleted of sodium. This, however, does not occur to a significant degree because the urinary sodium is replaced by other fixed cations, mostly calcium and potassium. The exact tubular mechanisms responsible for the hypercalciuria are not well understood.

The diagnosis is made on the biochemical findings and the administration of base will correct the acidosis and the hypercalciuria.

Fanconi Syndrome

In addition to the other well-known manifestations of this condition, such as glycosuria and amino-aciduria, hypercalciuria may also be present, since the increased excretion of organic acids causes a secondary increase in the urinary excretion of base. Milne *et al.*[20] suggest that there may in addition be defective reabsorption of $NaHCO_3$. If this is correct it would be an added factor in the production of hypercalciuria.

Primary Renal Hypercalciuria

After all the known causes have been excluded, there still remain cases in which the urinary output of calcium is above normal. These patients excrete a large amount of calcium in the urine for any given serum-calcium level and independent of the dietary calcium. It is patients in this group that have been labelled idiopathic hypercalciuria. It has been suggested that these patients have had staphylococcal pyelonephritis in the past.[5] It is possible to theorize along these lines and postulate resultant permanent tubular damage leading to defective reabsorption of calcium as an isolated phenomenon. There is, however, no evidence to support this view, nor has it been possible up to now to demonstrate any pathological lesion of the kidneys in these cases, either by means of renal-function studies or histologically.

A wide variety of single or multiple biochemical abnormalities have been described as a result of disordered function of the proximal renal tubule. In the classical Fanconi syndrome the triple resorptive defect of glucose, phosphate and amino acids occurs and the best-known single resorptive defect is renal glycosuria.

It seems possible that hypercalciuria exists within this group of conditions as a further example of a single resorptive defect and that patients exhibiting this abnormality are the ones now referred to as idiopathic hypercalciuria. With this concept of the underlying cause this term should be discarded and the term primary renal hypercalciuria applied to these cases instead. If used in its intended sense within the above classification, a clearer picture of the causes of hypercalciuria will emerge. Further investigation is required to substantiate this concept.

SUMMARY

In this article hypercalciuria is defined, a classification of the causes is presented, and observations are made on the diagnosis of the various causal conditions and the mode of production of hypercalciuria in these. The use of the term 'primary renal hypercalciuria' is suggested to replace 'idiopathic hypercalciuria'.

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HYDROMETROCOLPOS

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Hydrometrocolpos is a congenital condition in which the vagina and uterus are enormously distended by fluid secreted by the epithelium of the cervix, uterus and vagina, in the presence of an obstructing membrane or septum. A single acquired variety, due to vaginal stenosis (Markus cited by Morris[1]) occurs in old women.

CASE RECORDS

An African primigravida was admitted in labour; presentation vertex; foetal heart sounds slow, rhythm regular; cervix fully dilated and membranes had ruptured; meconium escaped copiously. One hour later a female baby was born, with the umbilical cord wound round the neck, and moderately asphyxiated. Cutaneous petechiae were seen in the baby's face next day. The baby's abdomen became increasingly distended during the next 4 days, interfering with respiration. There was no vomiting. The bowels acted normally. Micturition was normal. On examination there was a prominent bulge in the middle of the epigastrium with overlying skin veins. This swelling was firm and immobile and its shelving borders were ill defined. The upper abdominal wall was fairly tightly stretched over the mass. Straight X-ray of the abdomen showed outline of a large cyst in the abdominal cavity pushing up the diaphragm (Fig. 1)—no opaque medium was used. Respiration was almost entirely thoracic, with increased rate and excursions.

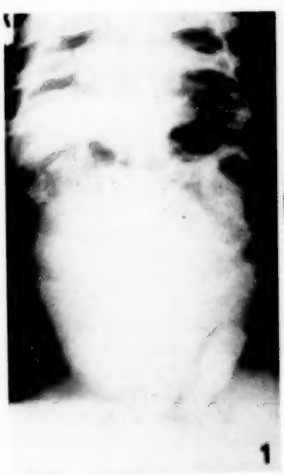

Fig. 1. X-ray of patient.

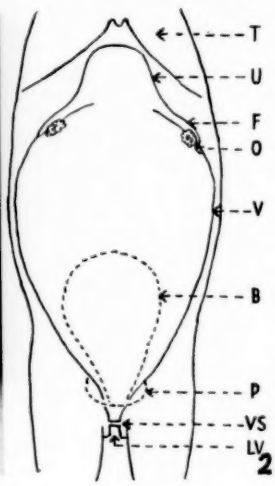

Fig. 2. Diagrammatic sketch of findings. T = thorax, U = uterus, F = fallopian tube, O = ovary, V = vagina, B = bladder, P = pelvic brim, VS = vaginal septum, L V = lower end of vagina.

Laparotomy on the 5th day (E.R.). Under local anaesthetic the abdomen was opened through a right paramedian incision. A large fusiform tumour was found, filling the peritoneal cavity. Tapering downwards it filled the pelvic brim and its base was fixed to the floor of the pelvis. The apex was formed by a relatively small anteverted uterus. The uterine tubes, on either side of the apical knob were diagnostic (Fig. 2). A mid-line incision was made in the anterior wall of the cystic mass, and about 100 ml. of fluid resembling liquor amnii was evacuated from the ballooned vagina. Vaginal examination located an accluding septum about 1 cm. behind the hymen. The septum was first palpated bimanually from above and below, and then punctured with artery forceps. A catheter was passed from the vulva and tied in. The abdominal vagina and the abdominal wound were then closed without drainage.

After the operation 200 ml. of blood was given intra-tibially. Crystalline penicillin, 50,000 units 6 hourly, and streptomycin, 5 mg. 12 hourly, were given intramuscularly for 6 days. The vaginal catheter was removed on the 4th day. Further courses of antibiotics were continued.

On the 17th day partial separation of the wound edges required secondary suture. The baby had bouts of vomiting and diarrhoea and intermittent pyrexia.

The abdominal swelling had subsided rapidly and soon became impalpable. A subsequent attempt to reinsert the catheter failed.

Subcutaneous infusion of electrolytic fluids were maintained as required.

The baby, however, eventually lost weight, developed cellulitis of the thighs and face, and died in a state of inanition at the age of 69 days. There were no other congenital abnormalities noted in this case.

DISCUSSION

Incidence

Most text-books and encyclopaedias do not even mention the name Hydrometrocolpos or Hydrocolpos. Gross,[2] however, described the condition in his *Surgery of Infancy and Childhood* under the heading 'Diseases of the Genital Tract' and Edith Potter[3] in her *Pathology of the Fetus and Newborn.*

Commandeur,[4] in 1904, described 1 case and reviewed 9 others, and Spencer[5] recorded 1 case in 1916. In 1940, after a silent gap of 24 years in the literature, Mahoney and Chamberlain[6] described 3 cases of their own, and Kereszturi,[7] recorded 1 case. In the next year Bowen[8] and Althoff[9] each described a case. Subsequently Rosenblatt and Wooley,[10] Morris,[1] Maliphant,[11] Sen[12] and Brews[13] each described a single case. We have therefore been able to trace 22 cases in all.

Age Incidence

Hydrometrocolpos occurs at two age periods, viz. neonatally and in the prepubertal period. Sen[12] found that the neonatal cases ranged from 1 day to 11 weeks old. In the prepubertal group, Althoff,[9] Bowen[8] and Brews[13] detected their cases at the ages of 14, 12 and 15 years respectively.

Pathology

The condition results from complete vaginal obstruction, This can be caused by an imperforate hymen or by vaginal atresia at a higher level forming a septum or retrohymenal membrane. The septum has been described by Mahoney and Chamberlain,[6] and Bonnet;[14] it was present in our case.

Excessive secretion of mucoid fluid takes place from the genital glandular tissue, mainly the cervical glands. It is known that in the presence of complete vaginal obstruction, normal secretion does not usually produce hydrometrocolpos. The current theory is that the excessive secretion is the result of raised concentration of maternal oestrogens in the foetal circulation; the absence of uterine bleeding is explained on the basis that the lining of the genital tract includes vascular and glandular components which may react independently or simultaneously to oestrogen stimulation according to their reactivity to hormonal stimulation. According to Sen[12] this theory has been confirmed by Markee (1936), Daron (1936), and Kaiser (1948).

During foetal and neonatal life the cervix is larger than the body of the uterus and probably secretes more actively. Both the cervix and body show increased secretory function in response to maternal oestrogens,[15] the vaginal epithelium matures, thickens and becomes keratinized, and the vaginal secretion becomes acid. The vaginal epithelial cells contain glycogen. Sen[12] points out that the same changes also occur in the prepubertal stage, thus coinciding with both the age period at which hydrometrocolpos is known to occur.

Mahoney and Chamberlain[6] confirmed histologically that there was increased activity of uterine glands and maturation of vaginal epithelium in their cases.

The vagina of the newborn is about 1-2 cm. long and 5-6 mm. wide (Gross). In hydrometrocolpos the anatomy of the genital tract is so grossly distorted that it may be unrecognizable unless the uterine tubes and ovaries are identified at the apex of the mass. The contained fluid is usually milky and 'chylous', but may be clear like liquor amnii. The distended vagina displaces the bladder forwards and upwards. The urethra and bladder neck are stretched and may be compressed. Kereszturi's case developed urinary retention 1 month old, laparotomy revealed the cause. Pressure on the uretero-vesical junction may produce bilateral hydronephrosis.[1,7] Infection further complicated Kereszturi's case, causing pyelonephritis.

Morris's case,[1] 7 weeks old, developed dysuria and constipation. There was also occasional blood in the stool. Intussusception was diagnosed; laparotomy revealed the unexpected cause. In this case there was also extensive oedema of the lower half of the body, caused by obstruction of the large veins. Morris emphasizes that hydrometrocolpos may remain undiagnosed until puberty, only to appear clinically as haematocolpos. Althoff,[9] Bowen[5] and Brews[13] detected their cases only a few months before the onset of menstruation.

Brews's case was 15 years old when she developed acute retention of urine. She appeared to have a classical, fairly large haematocolpos. Incision of the bulging vaginal membrane released 40 oz. of creamy white fluid. The fluid was sterile, and contained a few leucocytes. She menstruated normally 6 months later. Brews asks: 'Does this fluid represent a form of so-called "white menstruation" where the endometrium is not being subjected to, or does not respond to the influence of, a haemorrhagic factor, or does it represent an accumulation of non-menstrual fluid'?

Embryology

The Vaginal Septum. During the development of the embryo the paramesonephric ducts meet and fuse in the urogenital septum, producing the uterovaginal canal. The tip of this canal forms the soild vaginal cord. This cord unites with the sinovaginal bulbs, forming the vaginal plate. This plate is then canalized by the uterovaginal canal while the fused sinovaginal bulbs break down from below. According to Koff,[16] the lower 1/5th of the vagina is formed by the sinovaginal bulbs. It seems likely that the abnormal vaginal septum is due to failure of canalization at the level of the vaginal plate.

Imperforate Hymen. According to Koff's view[16] of the formation of the lower vagina, the hymen is a persisting membrane between the canalized bulbs and the urogenital sinus.

Clinical Picture

Reviewing 11 cases, Sen[12] found urinary retention in 2, abdominal tumour in 8, intestinal obstruction in 2. His own case and 2 others presented a vaginal bulge which became more prominent when the baby cried. Sen's case also had an imperforate anus. Respiratory distress was a feature in our case. In the prepubertal group backache and abdominal pain have been recorded.

Diagnosis

1. There is a swelling in the midline of the abdomen, arising from the pelvic floor. Rectal examination is essential.

2. Vaginal examination will demonstrate the imperforate vagina or vaginal septum. There may be an obvious bulge in the vagina when the baby cries. In the present case a bulge was not seen.

3. Puncture of the bulging membrane will release fluid similar to that following 'rupture of the membranes'.

4. Radiography was employed by Mahoney and Chamberlain,[6] and by Rosenblatt and Woolley.[10] Diodrast was injected into the hymenal bulge and the diagnosis was verified pre-operatively.

Differential Diagnosis

Morris[1] emphasizes that hydrometrocolpos is a surgical emergency deserving a place in the usual list of 'acute abdomens'.

When a hypogastric swelling is present, a distended bladder (e.g. due to congenital urethral obstruction such as urethral valves) must be excluded; urachal cyst must also be excluded.

Intussusception was considered in Morris's case.

In the epigastrium one may consider the obstructive group, e.g. congenital hypertrophic pylotic stenosis, duodenal stenosis or atresia, annular pancreas, malrotation and duplication of gut segments and their possible sequelae.

In the neonatal group other intra-abdominal swellings that may present in the mid-line are mesenteric cysts, teratoma and sympatheticoblastoma.

In the prepubertal group haematocolpos will be suspected. Hydatid cyst must be borne in mind.

TREATMENT

As a safety measure a needle is introduced and fluid is aspirated before a cruciate incision is made in the membrane. In some cases the obstructing membrane is less conspicuous and, owing to the proximity of vital structures, an abdomino-

perineal operation may be advisable. Then bimanual digital palpation should guide the perforating instrument safely between the urethra and the rectum. A catheter is inserted into the vagina and drainage should be maintained, for weeks or months if necessary, in order to ensure patency of the membrane.[2] Antibiotics may have to be administered over long periods.

Prognosis

This depends on the severity of the pressure effects. Sen[12] quotes a case who died on the first day of life from intestinal obstruction.

Early diagnosis and simple drainage play an important part in the prognosis.

Morris's case[1] underwent laparotomy, and then developed rupture of the abdominal wound and faecal fistula and died. One of Mahoney and Chamberlain's cases[6] underwent panhysterectomy and died.

Intercurrent infection is a great danger. Kereszturi's case[7] developed pyelonephritis, ending in death.

SUMMARY

1. A case of hydrometrocolpos in an African infant is recorded.
2. Available literature has been reviewed.
3. A plea is made for the wider recognition of this acute abdominal emergency.

We thank Dr. G. R. McLeish, M.D., Chief Medical Officer of the Wankie Colliery Hospital, for permission to publish this case; and Prof. James Louw of Cape Town for drawing our attention to Sen's article.

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THE ROLE OF THE ANAESTHETIST IN ECLAMPSIA *

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The aetiology of eclampsia remains obscure, and the methods of treatment controversial, so that it may seem presumptuous for an anaesthetist to become involved in such a thorny problem. Yet anaesthetics have been invoked as an essential part of the treatment of eclampsia since the birth of modern anaesthesia. Channing[1] in the U.S.A. (1848) and Simpson[2] in Scotland (1849) both recommended chloroform as a means of controlling fits, while Braun[3] in Vienna wrote extensively on the subject, publishing his findings in 1857, which afterwards appeared in a monograph form under the title 'Uraemic Convulsions'.[3] This title gives a clue to the features of the condition which were then considered important. Today, disturbance of renal function and convulsions are regarded as seriously as ever, but hypertension claims an even greater attention.

Essentially all forms of treatment aim at reducing the blood pressure, avoiding fits, and preventing renal damage. Ultimately, this implies the prevention of oxygen lack in any system of the body, and by no means of less importance, in the foetus. How far modern treatment succeeds in achieving these aims must be open to question since every year brings a new method. Perhaps this should be interpreted as a struggle for perfection, although more often than not it implies dissatisfaction with existing results, not only in terms of the ultimate outcome for mother and foetus, but also in terms of maternal morbidity and length of hospitalization.

In almost every case, sedation is the method of choice for lowering the blood pressure. This implies loss of consciousness, foetal depression and a diminished renal filtration pressure, since vasospasm has not necessarily been relieved. All three have obvious consequences, of which the anaesthetist no less than the obstetrician is fully aware. In an effort to avoid these hazards, at least in part, continuous conduction analgesia has been used with some measure of success.

RATIONALE OF THE METHOD

The rationale of this approach is simple. It is based on the assumption that all the ills that beset an eclamptic patient are the direct consequence of a high blood pressure. If that blood pressure can be reduced, as it can be in any subject by an extensive local analgesic block, then the following results may be expected, viz. (1) Prevention of fits, which are merely a symptom of a hypertensive encephalopathy, (2) the removal of a circulatory load from the heart, and (3) the alleviation of spasm in the renal vessels, which will allow filtration to occur at a lower pressure.

Further, these effects can be produced without rendering the patient unconscious, and without causing foetal depression. Whether the hypertension is the cause or effect of eclampsia may be disregarded since, happily, delivery

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makes the condition virtually self-limiting. The object is therefore to establish as far as possible an artificially normal physiology until this desirable state is effected more permanently by the body itself.

As a means of relieving anuria from other causes, splanchnic and spinal blocks have been employed since 1923,[4] but as far as can be ascertained, Paramore[5] was the first to try spinal analgesia as a specific treatment for eclampsia. However, it was not until spinal and extradural blocks could be maintained continuously that such a method became practicable.[6] The same remarks apply equally to the use of local analgesia as a means of relieving the pain of labour, although the value of doing so, especially in patients suffering from cardiac or respiratory disease, was recognized by several workers nearly 30 years ago.[7,8] Thus it was not until 1949[9] that a satisfactory technique was developed and tried which overcame the earlier difficulties. But already Hingson and others[10] had reported good results in 74 cases, claiming a 3·9% maternal mortality rate. This compares favourably with other methods; for example, the Queen Charlotte Hospital in London reports a 7% maternal, and a 23% foetal mortality rate when heavy sedation with morphine, chloral and paraldehyde forms the basis of treatment.[11] The first case of eclampsia to be treated with continuous spinal block in the United Kingdom was reported in 1949[12] and gave grounds for some encouragement. Since that time other cases have received similar treatment, but without exception all have been gravely ill and have failed to respond to more conservative measures. Such material can hardly be expected to provide a dramatic success rate, although in fact there was only one maternal death in a series of 10 cases, and that occurred from an extraneous cause a week after the cessation of treatment and at a time when it was felt that recovery was assured.

All too often it is forgotten that an essential feature of eclampsia is fits. But these in themselves may only represent a response to a variety of insults in a person predisposed to convulsions of any type.[13] They usually succeed in evoking some form of vigorous intervention, an anomalous situation of bolting the stable door after the horse or, in this particular case, the blood pressure has been allowed to run riot. It would seem that the rising blood pressure may 'trigger off' the convulsion, but in the meantime it may have led, surreptitiously perhaps, to other injuries, of which cerebral haemorrhage, cortical necrosis of the kidney, heart failure, pulmonary oedema and placental insufficiency are the most catastrophic.[14]

This is not an *apologia* for an unsatisfactory form of treatment, but rather an explanation of the problems which must be faced in the gravely ill eclamptic patient, for it is probably in such cases that continuous local analgesia is most useful, since the inherent risks of the procedure *may not* be justifiable in the mild case which responds to conservative measures. I stress the words *may not* because, although the treatment will be adequate, it is not always possible to provide the perfect conditions required if safety and success are to be assured.

ANALGESIC TECHNIQUE

Spinal, caudal and lumbar extradural blocks offer a choice of three routes by which continuous conduction analgesia may be maintained. Of these, the caudal approach is the least satisfactory since, as also when it is used as a form of analgesia in normal obstetrics, there is rarely a significant effect on the blood pressure.[15] Thus the choice lies between spinal and lumbar extradural analgesia. In both, a length of plastic tubing is inserted through a Tuohy needle, so that it may be conveniently directed into the subarachnoid or extradural space, and the needle then removed. The site of puncture is carefully sealed with paraffin gauze impregnated with penicillin to minimize the chances of infection spreading along the track of the tubing. The remainder or proximal part of the tubing is fastened to the patient's back by a length of adhesive strapping, so that the free end, to which a syringe remains attached, can be brought over the shoulder or placed under the pillow. An injection of local analgesic solution may then be made whenever desired. Lignocaine (xylocaine), 1% is the most universally useful solution, and the volume injected will depend on the individual response of the patient. Initially 2-3 ml. should be tried if the injection is intrathecal, and 15-20 ml. if it is extradural. Thereafter the dose may be varied in accordance with the needs of the patient and should be repeated whenever the blood pressure starts to rise again.

Response to Analgesia

The fall of blood pressure will depend on the level of analgesia achieved; but the blood pressure rather than the analgesia must determine whether or not the block is sufficiently extensive. Once the blood pressure has been controlled, sedation is no longer necessary to prevent convulsions, and the routine nursing care of the patient becomes easier. However, even greater attention to blood pressure, pulse rate and pressure is needed, and these must be charted graphically for rapid reference. It has been suggested that the establishment of a conduction block will increase the urinary output immediately, and this can be further aided by causing the blood pressure to 'swing'. In our experience this has not been the case, and the daily urine secretion by normal obstetric patients, eclamptics treated with heavy sedation, and eclamptics treated with continuous conduction analgesia, have been almost identical. In all instances the output increased after delivery had taken place unless permanent renal damage was present. Attempts to make the blood pressure 'swing' were not only ineffective in this respect, but tended to precipitate heart failure. Warning of this is given by a progressive fall in the pulse pressure and a rise in the pulse rate.[16]

As the time for delivery approaches, the blood pressure becomes more labile and it is increasingly difficult to control. The previous level of analgesia must often be increased, and injections made more frequently and with larger volumes of solution.

Complications of the Method

By far the commonest complications that will be encountered during the management of a patient under continuous conduction analgesia are hypotension and paralysis of the muscles of respiration. Thus at all times the means to combat respiratory depression, overdosage with the local analgesic, and too violent falls in blood pressure, must be immediately available. In addition, since sphincters will be paralysed, regular emptying of the bladder and rectum become necessary, and measures must be taken to guard against such peripheral injuries as foot-drop.

But the greatest hazard of all, and the one which must be taken into account before deciding on this form of treatment, is infection. No efforts must be spared in trying to avoid this danger, and unless the sterility of the initial technique can be guaranteed, the method is absolutely contra-indicated. Precautions to prevent the spread of the infection along the track of the tubing have already been mentioned, and this is of very real importance when the patient's back and bedding may become contaminated with liquor or faeces; and finally—and this is a difficult technical problem—the prevention of contamination of the free end of tubing which might result in organisms being pumped into the patient by subsequent injections.

Comparison of Spinal and Extradural Analgesia

Before comparing these two approaches one must assume that there will be no overriding technical difficulty in establishing either the one or the other. Both types of block are capable of lowering the blood pressure efficiently, although the results of an extradural injection take rather longer to become apparent. Also, after about 24 hours, it will be found that the volume of solution to be injected into the extradural space must be progressively increased and there is thus a risk of toxic reactions from overdosage with the local analgesic.

An extradural abscess is considered less seriously than meningitis, so that once more the risk of potential infection may become the overriding factor in the choice of methods. Neither complication can be viewed with complacency but, since extradural analgesia can be expected to give as good results as spinal analgesia with a slightly greater safety factor, it should be considered the method of choice. In some instances, however, when treatment must be maintained for more than 24 hours, extradural analgesia may become less effective in controlling the blood pressure. If this happens, the extradural catheter can be removed and continuous spinal analgesia established instead. But such a change should not be undertaken lightly, for now the subarachnoid tap must be made through a potentially infected extradural space.

CONCLUSIONS

The establishment of anaesthesia for operative delivery in eclampsia or for the immediate control of convulsions has not been discussed, because the difficulties are reasonably clear cut. No one doubts the existence of these problems, yet it is not easy to find a solution, or even to suggest a routine, which at the same time will avoid undue foetal depression, prevent the aspiration of stomach contents, and on occasion provide deep anaesthesia, albeit for a short space of time. Almost any anaesthetic technique can be satisfactory, but the really important factor is the competence of the anaesthetist. On this point there can be little disagreement.

In describing the technique of continuous conduction analgesia I have attempted to suggest a way in which the skilled anaesthetist may be of service to the obstetrician in the gravely ill patient. It is not suggested that the method should be applied indiscriminately since it carries risks which cannot be accepted for mild degrees of eclampsia. However, when the situation is sufficiently grave and the life, not only of the foetus but of the mother also is at stake, then the relative merits of continuous conduction analgesia can be viewed in their proper perspective. By calling on the anaesthetist at such a time the obstetrician may perhaps save the life of his patient, besides sparing himself much mental anguish.

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CIRCULATORY CHANGES IN ANAESTHESIA*

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The advent of modern anaesthesia has seen more than just the introduction of new drugs and techniques, for it has created a general desire in the anaesthetist to learn more about the physiological changes occurring in a patient undergoing surgery.

* A paper presented at the South African Medical Congress, Durban, September 1957.

In England little over 10 years ago the anaesthetic agent of choice for most surgical cases was ether. Today the ether bottle stands on the shelf gathering dust. This rapid transformation has been brought about because we now know that we can provide better conditions for the surgeon with less risk to the patient by combining the use of different drugs. A start has been made on the road to learning some of the changes that take place in the circulation of the

anaesthetized patient, but in the past this field has been so neglected that almost the whole physiological basis of the unconscious patient awaits discovery.

BLEEDING IN SURGERY

The rapid change-over from the era of ether to that of the muscle relaxant brought with it many problems, but foremost amongst these was the observation that the skin and muscle tissues showed increased oozing during operation. Plethysmography—that is the measurement of blood flow—revealed that there was an enormously increased flow of blood through both the skin and muscle tissues immediately a state of unconsciousness was induced. Experimental evidence suggested that the cause of this generalized vasodilatation was a temporary suppression of vasomotor activity. If the level of anaesthesia was kept constant, the blood flow returned to the normal level after about 40 minutes to 1 hour. It was found, however, that altering the depth of either ether or cyclopropane anaesthesia produced a profound change in the blood flow to the tissues. Thus, as the depth of anaesthesia increased so the blood flow diminished. This action is believed to be a local rather than a central one and to be mediated through either a direct effect of the anaesthetic substance on the vessel wall or the liberation of some constrictor substance.

The net result of these findings helps to explain why there is less bleeding in the presence of deep ether or cyclopropane anaesthesia than there is during light anaesthesia combined with a muscle relaxant.

Measures used to diminish Bleeding.

The problem of oozing from the surgical wound is clear. Prevention, however, is not so easy. *Deep anaesthesia*, employing toxic doses of anaesthetic agents, is effective, but this in turn leads to other complications. The anaesthetist, therefore, has tended to look around for some other suitable means of reducing 'anaesthetic ooze'. Local infiltration with a dilute solution of adrenaline in saline (1:250,000) is effective. Similarly, alterations in the carbon-dioxide tension of the blood influence the degree of capillary bleeding. A raised carbon-dioxide tension not only helps to raise the blood pressure and increase the cardiac output but also has a direct dilating action on the peripheral vessels. On the other hand, *hyperventilation* combined with carbon-dioxide absorption lowers the tension in the blood and reduces bleeding. Some anaesthetists in their enthusiasm for overbreathing the patient sometimes create a positive pressure within the chest during both phases of respiration. The result is that the flow of blood into the thorax is impeded and the cardiac output and the blood pressure fall. Although a positive intrathoracic pressure during expiration does help to reduce bleeding, the effects are so grossly unphysiological that it would not be used in clinical practice.

An outstanding advance in the reduction of bleeding in surgery was the re-discovery of the importance of *posture*. In the horizontal position none of the advantages of collapsed venous channels and capillaries can be obtained. The simple expedient of tilting the body 10°-15° from the horizontal in cranial, thyroid or certain plastic operations is often sufficient to curtail major oozing. Even under anaesthesia, however, the patient is still able to compensate for alterations in posture, and thus the blood pressure and cardiac output should be unaffected by this manoeuvre. Temporary interruption of this compensatory mechanism will seriously reduce the systolic pressure and consequently the bleeding in the upper half of the body. The desire for a relatively 'bloodless field' has led anaesthetists to search for satisfactory and safe means of producing these conditions. One of the ways of achieving them is to administer a *drug* capable of producing *peripheral vasodilatation* and to combine this with suitable posture so that the major part of the blood volume accumulates in the dependent part of the body, leaving the other half relatively dry. In short, the patient is bled into his own tissues. Nearly all the tranquillizing drugs are vosodilators, and one of the most potent is chlorpromazine. Intra-arterial injections of this drug have shown that it is very effective in producing a local vasodilatation which persists for 2-3 hours (Foster *et al*, 1954). Very large doses of these drugs are capable of producing some degree of block of autonomic activity but the principal action on the circulatory system is one of local dilatation.

Many authors have been tempted to claim that these drugs prevent surgical shock, and in so doing have created the impression that such an action is necessarily a beneficial one. If one concedes that the 'shock reflex', is merely a generalized vasoconstriction in response to haemorrhage or trauma, then it is certainly true to say that these drugs prevent this vasoconstriction from occurring. But if the signs of shock are merely an effort on the part of the body to guard against loss of body fluids, can anyone say that destruction of this line of defence is necessarily beneficial? It is a well-known fact that patients under the influence of vasodilator drugs tolerate blood loss very badly, and in the absence of a constrictor reflex even a small haemorrhage may prove fatal.

The quest for a greater control over the degree of blood during surgery has led to the introduction of the *ganglion-blocking drugs*. Opinion is strongly divided on the risks of using these drugs in clinical anaesthesia. At St. Thomas's Hospital in London we adopt a somewhat middle-of-the-road attitude and believe that in certain circumstances they have a very useful part to play. Thiophanium (Arfonad) combined with posture is the most frequently used. This technique is reserved for cases likely to suffer severe blood loss, such as block dissection of the neck, prostatectomy, Wertheim hysterectomy, and certain plastic procedures. The head-down tilt is favoured and the duration of hypotension is limited to the minimum time necessary for beneficial results. The presence of any signs of myocardial or cerebral ischaemia is taken as the only absolute contra-indication to the use of the technique; severe hypertension does not *per se* appear to lead to a harmful effect. As a safety factor it is of paramount importance that the systolic pressure should not fall lower than 70 mm. Hg. for more than a few minutes and under normal conditions the pressure is controlled around 80 mm. Hg. In the majority of cases this level gives very satisfactory operating conditions. Any blood loss must be instantly replaced by transfusion.

It must be stressed that rapid alterations in posture in the anaesthetized patient are dangerous, particularly in the presence of autonomic blockade. Whatever the angle, blood tends to accumulate in the most dependent part and a sudden alteration in position may lead to a fatal hypotension. Such an action is particularly severe in the absence of any powers of vasoconstriction. As a precaution against any

such change in the circulation, any patient who has been submitted to a severe degree of tilt during operation is returned to the recovery room on a tilting trolley and only gradually, over a period of 2-3 hours after operation, is he returned to the horizontal position—and this only if the readings of pulse and blood pressure are within normal limits.

Haemorrhage and anaesthesia

That loss of blood leads to generalized vasoconstriction has been known for many years, but only recently was it deliberately adopted as a clinical practice for patients undergoing cranial surgery, in order to reduce bleeding and diminish the size of the brain. The technique was alternately to remove and replace blood from the radial artery to keep the systolic blood pressure at a level of 80 mm. Hg (Jackson, 1954). Though this practice never attained wide popularity, it stimulated other workers to study the effects of haemorrhage in the anaesthetized patient. One of the principal findings was that it was possible suddenly to remove up to 25% of the estimated total circulating blood volume without any significant change occurring in either pulse rate or blood pressure. Such a finding suggests that many patients may unwittingly be returned to the ward in a state of apparently normal circulatory balance yet, in reality, be suffering from severe hypovolaemia (depleted blood volume). Many of these cases show a marked hypotension and tachycardia in the immediate post-operative period.

Measurement of blood loss during surgery

The anaesthetist would clearly welcome a quick and simple means of assessing blood volume both before and after the induction of anaesthesia, but it will probably be many years before such a piece of apparatus is available. The simple expedient of weighing swabs, however, is easy to manipulate. A commercial spring-balance normally used for letters and light parcels is obtained and the scale is graduated, so that a zero reading represents the weight of 6 small, 3 medium and 1 large swab. The exact relationship in numbers must be determined at the outset. The scale is then graduated in millilitres of blood. In use, the nurse merely places the appropriate number of swabs on the scale and records the amount of blood present before proceeding to arrange the swabs. If a suction bottle is used this is also graduated in millilitres of blood. The surgeon must pay particular attention not to add fluid from extraneous sources, such as cysts, etc., to the total assessment. Some method of assessing blood loss in all cases of major surgery is essential, and at the present time the swab-weighing technique appears to assess the circulatory state of the patient with reasonable accuracy for clinical purposes.

SURGICAL SHOCK, STRESS AND ANAESTHESIA

The term 'shock' has probably led to more confusion of thought than almost any other in medicine today. Crile visualized surgical shock as a state occurring in the brain due to the constant bombardment of the brain by stimuli from the surgeon's knife. On the other hand there was the severity of the stimulus to be considered, and on the other the protection of the brain that was achieved by deepening the anaesthesia. If the degree of stimulus was too great for a particular depth of anaesthesia then a state of surgical shock supervened. The advent of d-tubocurarine has made this theory untenable, for we now know that it is possible to keep a patient in the lightest possible state of anaesthesia and yet perform the most traumatic operations without any signs of the development of surgical shock. It is most unlikely that this protection has anything to do with a specific action of curare on the brain or autonomic ganglia, as other relaxants with very different actions also offer this protection. I believe the shock syndrome is almost entirely due to alterations in blood volume, e.g. haemorrhage and dehydration. Rough handling of the viscera in a partially paralysed patient may be sufficient to induce a state of bronchoconstriction in the lungs and a tightening in the thoracic muscles, and this in turn may produce marked alterations in the pulse rate and systemic pressure. These changes are, however, rare in the completely paralysed patient on controlled repiration. Clearly certain visceral reflex pathways do exist, but we should not use the knowledge of their presence to explain errors of our own technique.

It is now almost universally accepted that all patients undergoing major surgery display the typical signs of the stress syndrome in the post-operative period—that is to say, they develop oliguria, sodium retention, leucopenia, etc. However, in 1955, Flear and Ruscoe Clarke showed in an experimental study in man that the degree of these reactions can be markedly reduced, or even abolished, with adequate transfusion therapy. It would be seen, therefore, that in the prevention of both 'surgical shock' and 'stress' it is most important to pay particular attenion to even minor alterattions in blood volume during surgery.

THE ARRESTED CIRCULATION

The pressing demands of cardiac surgery for a completely 'dry field' are such that it is necessary to perfect some means whereby the heart can be omitted from the circulation for an hour or so without causing any damage to the vital organs. *Hypothermia*, by lowering the tissue metabolism, has offered one approach to this problem. I think it would be true to say that it has not lived up to the original expectations, but many successes have been achieved with this method. It is now generally agreed that it is unsafe to lower a patient's body temperature below 30°C (normal 37°C), and this level permits the surgeon only a very limited time to perform his repair (about 10 minutes). If, however, the danger of increased irritability of the heart at low temperatures could be removed, then the value of this technique would be enormously increased.

At the present time the most promising line of approach is by the use of a *machine* that will take over the work of the heart and lungs. But this alone raises many interesting problems of physiology, and offers an exciting new field of development, in which the anaesthetist will be required to play his part.

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SOUTH AFRICAN MEDICAL CONGRESS, DURBAN 1957

This, the 41st South African Medical Congress and (to pursue a curious distinction) the 20th Annual Scientific Meeting, took place in Durban from Sunday to Saturday, 15-21 September 1957. The Congress was planned on broad and ambitious lines, which achieved a success that must be a source of deep satisfaction to its promoters, to whom the thanks of the Medical Association of South Africa are due. About 1,000 members of Congress registered, in addition to 500 associate members (chiefly members' wives and relatives). The City of Durban provided a lovely setting for the proceedings. The weather was changeable, some days being bright and sunny and others cloudy and overcast; it was ideal for such an occasion, never being oppressively hot. The week ended with a heavy thunderstorm on the Friday night which was over by Saturday morning. The Durban newspapers reflected great public interest in the Congress and devoted many columns to reporting and commenting on its proceedings.

A *Congress Brochure* was presented to all members of Congress, comprising 204 pages, of which all but 27 consisted of Congress matter, with about 30 illustrations, including 24 portraits of Congress officials and others. This publication was edited by Dr. F. B. Proksch with the assistance of Dr. J. J. Pauw. It contains messages from His Excellency the Hon. E. G. Jansen, Governor General of the Union, His Worship the Mayor of Durban (Councillor Percy Osborn) and the President of the Medical Association of South Africa (Dr. H. Grant-Whyte); detailed information concerning the Congress, including the Trades Exhibition, the Scientific Exhibition and the Arts Exhibition (Doctors' Hobbies); interesting articles on Durban and its history (Dr. Proksch), the Natal hospitals (Dr. R. E. Stevenson), the Natal University medical school (R. S. McK. Thomson) and the College of Physicians and Surgeons of South Africa (Prof. G. A. Elliott); and an article by Dr. J. Drummond on 'A Decade of Medicine'; together with synopses of 121 of the 200 papers presented at the plenary and sectional sessions. Each member was also supplied with a pocket diary of Congress and the associate ladies with a similar small diary of those Congress events which were designed or were available for them.

The tone of the proceedings was set at a high level, for which the medical profession of South Africa and the Association are greatly indebted to the many visitors from the UK, the USA and elsewhere, who contributed notable addresses, and to the high quality of the contributions of South African members of Congress.

The Congress Headquarters and the Trades Exhibition were located at Red Cross Hall, Old Fort Road, Durban. The plenary and sectional sessions and most of the Group meetings were also held in Red Cross Hall and in buildings in close proximity to it, viz. Haining Hall (M.O.T.H.), the B.E.S.L. Hall, the Boy Scouts' Hall and the St. John Hall. The Scientific Exhibition was held in the Old St. John Hall, Epsom Road, Durban, and the Arts Exhibition (Doctors' Hobbies) in the Art Gallery, Durban Town Hall.

Public Lecture. Congress proceedings began on Sunday evening 15 September with a public lecture by Dr. T. C. Routley, C.B.E., Past President of the Canadian Medical Association and the British Medical Association. This was held at the Playhouse Cinema Theatre, and was attended by an audience of many hundreds. The title of the lecture was 'You, and your Doctor', and it dealt mainly with the aims and achievements of the World Health Organization and the World Medical Association, of which latter Dr. T. C. Routley is Consultant-General. A report of the lecture will be published in a future issue of the *Journal*.

Openings of the Exhibitions. Monday morning saw the official openings of the Trades Exhibition, the Scientific Exhibition and the Arts Exhibition, followed in the afternoon by the first plenary session.

Congress Opening Ceremony

On Monday evening at 8.30 the opening ceremony took place in the Durban City Hall. It was an impressive and colourful occasion. Academic dress was worn, and the large hall was well filled by a great gathering of Congress members and others. After a musical programme on the organ played by Mr. Errol Slatter, L.R.S.M., the distinguished platform party entered in procession with musical honours and preceded by the Association mace, and the chair was taken by the President of the Medical Association of South Africa (Dr. H. Grant-Whyte).

His Honour the Administrator of Natal (Mr. D. G. Shepstone), Chancellor of the University of Natal, delivered an address and formally declared the Congress open.

Dr. J. S. du Toit, retiring President of the Association, was invested with the insignia of Past President; Dr. A. W. S. Sichel, who the previous week had vacated the office of Chairman of Federal Council, with the insignia of Past Chairman; and Mrs. H. Grant-Whyte with the Badge of Office of President's Lady.

Next the Association's Bronze Medal for distinguished service to the medical profession was presented by the President to the following recipients, after the respective citations had been read by Dr. A. H. Tonkin, Secretary of the Medical Association: Dr. B. A. Armitage, Pietermaritzburg; Dr. A. Broomberg, Durban; Dr. C. M. Grundlingh, Pretoria; Dr. M. Shapiro, Johannesburg and Dr. R. Theron, Bloemfontein.

The President also awarded (*in absentia*) the Association's Hamilton-Maynard Memorial Medal to Dr. Geoffrey Dean, of Pretoria, and Leipoldt Memorial Medal to Dr. H. Braude, of Kroonstad.

Dr. H. Grant-Whyte then delivered the Presidential Address,* after which the platform party retired in procession.

Congress Banquet

The Banquet was held in the City Hall on Tuesday at 8.15 p.m. It was restricted to medical members of Congress, of whom over 600 sat down. This large attendance was easily accommodated in the magnificent City Hall which, beautifully decorated, presented a brilliant scene. Dr. H. Grant-Whyte presided. The toast of the Medical Association of South Africa was proposed by Dr. T. C. Routley, of Canada, and Dr. A. W. S. Sichel replied. Dr. A. Broomberg proposed the toast of the Guests, which was replied to by Sir Russell Brain, Bt., of London, and Prof. E. L. Bortz, of the University of Pennsylvania. Dr. T. Cawthorne, of London, Prof. N. M. Dott, C.B.E., of Edinburgh, Prof. V. Kinross Wright, of the Baylor University, Texas, and Dr. A. J. Wrigley, of London, also spoke. The eloquent speeches were much appreciated by the company and added greatly to the memorable character of the occasion.

Mayoral Reception

His Worship the Mayor of Durban (Councillor Percy Osborn) entertained members of Congress and their ladies to a Civic Reception in the City Hall on Wednesday at 6 p.m. A very large party enjoyed the Mayor's hospitality and were welcomed by his Worship in a very entertaining address. The next night (Thursday) the City Council invited Congress members and their ladies to the symphony concert at the City Hall (Durban Civic Orchestra, conductor Fritz Schuurman, and solo pianist Kendall Taylor).

Congress Ball

The concluding social event, a Ball in the City Hall, at which 600 couples attended, was another brilliant affair. The Hall was lavishly and artistically decorated with flowers, and the scene was one of beauty and gaiety.

In the course of the evening the Mayor of Durban, on behalf of the City Council, presented a plaque bearing the City arms to Dr. H. Grant-Whyte in commemoration of this outstanding Medical Congress. Dr. Grant-Whyte then made commemorative presentations to Dr. A. Broomberg (chairman of the Organizing Committee), Dr. B. Crowhurst Archer and Dr. S. Dislen (Joint Organizing Secretaries) and Dr. J. Kelman Drummond and Dr. A. J. Wilmot (Joint Medical Secretaries).

SCIENTIFIC MEETINGS

Four *plenary sessions* were held, on Monday, Tuesday, Thursday and Friday. They were attended by probably the largest audiences ever seen at the scientific meetings of a South African Medical Congress. The overseas members took a prominent part, and the contributions were of a high standard. The subjects of the symposia and the readers of the papers were as follows:

* Published in the *Journal* of 21 September 1957 (**31**, 945).

1. Cerebral Vascular Disease and the Problems of Aging: Sir Russell Brain (London), Prof. E. L. Bortz (University of Pensylvania), Dr. M. M. Suzman, Dr. F. H. Kooy, and Mr. K. Lewer Allen.

2. Parasitic Diseases of Man in Africa: Dr. M. Gelfand, O.B.E. (Salisbury, S. Rhodesia), Mr. C. Marks (ditto), Prof. Paul C. Beaver, and Dr. R. Elsdon-Dew.

3. The Surgery of Repair: Sir Harry Platt (Manchester), Mr. W. Gissane (Birmingham), Prof. T. Pomfret Kilner, C.B.E. (Oxford), Prof. Norman M. Dott, C.B.E. (Edinburgh), and Prof. J. H. Louw.

4. Recent Advances in Child Care: Prof. Alan Moncrieff, C.B.E. (London), Dr. D. M. T. Gairdner (Cambridge), and Dr. H. L. Wallace.

Some of the sectional meetings were also in the nature of symposia, for instance: (1) A combined meeting of the sections of Medicine and Surgery dealt with the subject of Thyrotoxicosis, and the following speakers contributed to the symposium: Dr. J. S. Richardson, M.V.O. (London), Prof. Paul Wilflingseder (Innsbrück), Mr. L. V. Pearson, and Dr. Maurice Weinbren; and (2) a meeting of the section of Neurology, Psychiatry and Neuro-surgery was devoted to a symposium on the Treatment of Pain, to which papers were contributed by Sir Russell Brain (London), Prof. V. Kinross Wright (Baylor University, Texas), Dr. J. S. Richardson (London), Dr. B. Crowhurst Archer, and Mr. A. Lewer Allen.

At the meetings of the 19 sections into which the scientific meetings were divided (many of them combined meetings of two or more sections) 163 papers were presented, making, with the 17 papers read at the plenary sessions, a total of 200 scientific papers presented to the Congress.

The national Groups of the Association held business meetings during the Congress.

TRADES EXHIBITION

As always at the Medical Congress, the Trades Exhibition constituted an important central feature of Congress. There were 47 exhibiting firms, from South Africa and other parts of the world, mostly manufacturers and distributors of drugs, instruments, infant and invalid foods, and medical publications. The stalls were staffed by a large number of well-informed representatives of the exhibiting firms. The exhibits showed the newest and most important products, and the relative therapeutic information was available to enquirers. Simplicity was the keynote of the exhibition, and there was little of the cluttering up of stalls with a multiplicity of 'lines' which was sometimes seen at the Trades Exhibitions of former Congresses. The exhibits were judiciously chosen and the exhibition as a whole presented an attractive appearance.

Dr. J. Stolp was the convener of the Trades Exhibition Subcommittee.

Dr. Grant-Whyte's Address

The Trades Exhibition was opened by the President, Dr. H. Grant-Whyte, who said that to no duty at this Congress had he looked forward with greater pleasure. He was one of those doctors who were always ready to listen to the pharmaceutical expert. The Trades Exhibition, he said, was always a central feature of their medical congresses, to which it was of considerable financial importance. He took this opportunity to thank the overseas members who had contributed so much to the interest of this Congress, and also the pharmaceutical houses who had subscribed so generously to the overseas-visitors fund.

This was only one instance of the financial assistance which the pharmaceutical houses gave to medicine. Vast sums were ploughed back into the pharmaceutical industry and research. These sums, of course, ultimately came out of the pockets of the public who paid for the drugs which these houses put on the market. But the relative importance in the national economy of what is spent on drugs was illustrated by the following comparative annual expenditure per head in the USA: Drugs £3 12s. 8d., alcoholic drinks £19 15s. 2d., tobacco £11 9s. 11d., and repair and maintenance of motor cars £6 9s. 4d.; 4% of the total national expenditure was on drugs. Pharmaceutical houses were playing a prominent part in the fantastic rate at which medicine was advancing.

Dr. Grant-Whyte referred to the fact that in the national service the doctor's freedom in prescription was limited by the Tender Board's insistence on the lowest price. In medicine, quality ought to be the main consideration. He expressed the thanks of the Association to the exhibitors and invited members to spend ample time in examining the exhibits and in conversation with the experts in charge.

Mr. M. Stabler, Chairman of the Medical Exhibitors Association, thanked Dr. Grant-Whyte. He expressed the exhibitors' appreciation of the way the Congress committee ha dco-operated in the organization of the exhibition. They associated themselves in the welcome to the overseas visitors. They welcomed all members of Congress to the exhibition and wished the Congress every success.

SCIENTIFIC EXHIBITION

At the Scientific Exhibition, which was held at the Old St. John Hall, Epsom Road, the main exhibition consisted of 41 sets of exhibits shown by University departments, other institutions and individual workers. It was of high medico-scientific value and well worth many hours of close study. Another section of the exhibition was devoted to medical instruments and books. Cinematograph films were shown throughout the week, dealing with various branches of medicine and surgery; 5 of them had been made by individual practitioners, and the rest by institutions and organizations.

This year's exhibition showed a considerable advance on its predecessors at former medical congresses, which presaged well for future developments.

The Chairman of the Scientific Exhibition Subcommittee was Mr. B. W. Franklin Bishop, and the hon. secretaries Dr. E. Rosenberg and Mr. G. Stafford Mayer.

The Scientific Exhibition was opened by Dr. E. G. Malherbe, Principal of the University of Natal, who remarked that it contained so much of educative value to the ordinary man and woman, and even to the high-school pupil, that it should not be limited to the medical profession, but should be kept open for the public. Dr. Malherbe's address is reported at page 1022 of this issue of the *Journal*.

ARTS EXHIBITION: DOCTORS' HOBBIES

The Hobbies Exhibition was a display of 'work' done by doctors as a relaxation from their professional duties, and by their wives and families. Some 75 exhibitors put up about 100 exhibits, many of great merit or interest. The largest section was the paintings, of which 86 were grouped in 28 exhibits. Amongst these one noticed specially 3 oils by Sybil Traut (Mrs. John Richardson, of London), and portraits in oils of Dr. J. Drummond and Mrs. Kelman Drummond by Dr. J. Drummond's daughter (Mrs. R. Yuill), and a portrait of Dr. H. Grant-Whyte by Mrs. Doreen Wynberg. Dr. Morris J. Cohen's exhibition of 129 pieces of shellcraft made a wonderful centre-piece to the exhibition, as his work always does. The classes of exhibits were of varied interest but were too numerous for mention, except perhaps the antique medical books and the medical curiosa. Dr. Cohen and his colleagues on the Subcommittee are to be congratulated on the great success of this exhibition.

Mrs. Richardson's Address

This Arts Exhibition was opened by Sybil Traut (Mrs. John Richardson, herself an artist (and an exhibitior in this exhibition) and the wife of one of the distinguished overseas visitors to the Congress. She said that almost all her life she had been concerned chiefly with artists and doctors; part of her training had been under Professor Tonks at the State School of Art, London, who was both a medical man and a great painter, as well as an inspiring teacher. Many doctors she had known had found their greatest pleasure in the arts, and their work convinced one of the sincerity and appreciation of beauty which they brought to it. This was hardly surprising since both professions trained one to look into the heart of things and try to understand them. With doctors, however, their work had a special quality in bringing them relief from the stresses of their lives. In the doctor-artist the word 'amateur' had its true meaning—not one who dabbles and is outpaced by the professional, but one who practices his art or craft for the love of it.

Mrs. Richardson assured her hearers that they would find much in the exhibition to convince them of this. Apart from the

paintings there was such a diversity of skills that it could never be suggested that doctors in South Africa had no interest besides their 'shop' or that their wives and families were only telephone drudges.

Mrs. Richardson expressed her thanks for 'the incredible kindness' which she and her husband, like all the visitors from overseas, had received, and in declaring the exhibition open she thanked Dr. Cohen specially for his personal kindness and congratulated him on his shell work which, she said, could not fail to find pride of place in an exhibition of this kind anywhere in the world; it had caused much admiring comment when seen on television in Britain last year. Mrs. Richardson also made special reference to the flowers which, she said, were arranged with an art that rivalled the exhibition itself.

THE DOCTOR, THE SCIENTIST AND THE PUBLIC

E. G. MALHERBE

Principal, University of Natal

In formally opening the Scientific Exhibition of the South African Medical Congress, Durban, 1957, Dr. Malherbe stressed the basic importance of the basic sciences for medicine. It was true that medicine was an art. The surgeon, for instance, needed the skill of the expert craftsman, and courage, as well as knowledge; and the handling of human beings and the intuitive side of diagnosis were an expression of this art. Nevertheless, medicine was becoming more and more of a science. This was true not only on account of its increasing dependence on physics, chemistry and biology but also in the strict use of scientific method in dealing with patients and disease. Scientific method involved the careful observation of facts and then the framing of a hypothesis or theory to explain these facts, according to which a remedy might be applied. The validity of a hypothesis depended on how carefully it was tested by control, and by using control groups. It was only by keeping crucial factors constant while observing the occurrence of certain other seemingly related phenomena that it was possible to determine whether their relationship was causal or was due to mere concomitance. As an example Dr. Malherbe referred to the theories advanced today in seeming conflict with one another about the relation between cigarette smoking and lung cancer, and between smoke in the atmosphere and cancer.

The Atomic Age

The importance for medical practice of a knowledge of the basic sciences had been demonstrated anew by the advent of the atomic age. The physicist had recently released forces which might have the most profound effects on human health and on life itself. We had long been familiar with coal and oil as a source of the primordially stored energy of solar radiation. This source of power was about twice as effective as the familiar wood fire. But now man was experimenting with a different kind of primordially stored energy. In rearranging not a few atoms but a few nuclei of atoms they were producing effects not two or three times that of the wood fire, but a million times greater. This million-fold factor had come in a period of a few years, and it was not surprising that people were worrying about the use of this new energy. The ineptness of explanation by scientists was the key to some of the hysteria that was at present affecting most thinking people in the world.

This was an illustration of the great problem of communication—communication not only between the scientist and the public but between scientists and medical men.

The scientists told them that a human being consisted of 10^{14} cells, all of which originated from one small cell. Everything they knew about life told them that the cell effected this separation and differentiation into different organs and different cell-systems in terms of large molecular systems, and biological research showed that these molecular systems had a cold integrity, a perfection of their own structure, their own pieces, their own past, which was passed on from cell generation to cell generation. In these molecules of nucleic acid not one atom might be out of place in the thousands of millions of atoms constituting them. Into this marvellously ordered structure radiation brought its devastating high energy releases, and we were told that when the track of one of the beta particles released in radio-activity went through a nucleic-acid molecule it broke it; and that the chance was low of its ever being re-formed as it was before. Nature was self-recuperative and self-regenerative, but in this particular case there might be a threshold beyond which regeneration was impossible; research workers in biophysics seemed to be convinced of the always-harmful effect of radiation on the basic code-determining form-producing molecules in the cell. This seemed to be most important when it came to affecting the stability of the human genes.

The present-day release of radiation by atomic tests was accompanied by a statistical increase in deaths—for example an increased incidence of deaths from leukaemia. Dr. Malherbe said he was not an expert on these aspects, but he wanted to stress the main general point, viz. that if scientists were going to release these gigantic forms of energy they had to make their medical thinking commensurate with it. Therefore a very close cooperation was needed between the medical men and the scientists. This combination was of course best achieved in one person; but it was seldom nowadays that a profound training in medicine was found combined in one person with an equally profound training in, say, biophysics.

The speaker then referred to the need for the scientific training of medical students, and expressed the opinion that training should be so organized that at least 10% of the students took an M.Sc. degree in science as well as a medical degree.

The Motor Car as Killer

Dr. Malherbe went on to observe that in this valuable exhibition there was no exhibit concerning one of the most potent killers they had around them—the motor car and the motor cycle. He could not see why orthopaedic surgeons and other medical men should not cooperate with designers of motor vehicles in order to lessen the great toll of injury inflicted on the human body in collisions which the outer body of the car survived. These injuries were exaggerated by unnecessary protruberance of gadgets and ornaments within the car. They should be realistic about the fact that people got knocked about within the vehicle when it came to a sudden stop. Why, therefore, should the designers not line the car with sponge-rubber cushioning and eliminate protruberances, so that what otherwise would be a mild shock should not be converted into serious injury. The speaker stressed this as an example of the need for cooperation between medical science and modern technology.

A Pedagogical Effort

In declaring the scientific exhibition open Dr. Malherbe said that it should not be limited to medical people to see. That was preaching to the converted. The exhibition contained so much of educative value to the ordinary man and woman—even for high-school pupils—that he felt that certain sections should remain open for them. The public should be shown what great organizations had been built up to deal with disease—its diagnosis, its cure and its prevention. This was an example of the importance of communication—a triangular communication between (1) the medical man, (2) the scientist and engineer and (3) the public.

PRESCRIPTIONS FOR HABIT-FORMING DRUGS

The Chief Regional Health Officer, Office of the Union Health Department, Durban, has addressed the following communication to the Natal Coastal Branch:

'It has come to the notice of this department that some medical practitioners endorse their prescriptions for habit-forming drugs with the statement *to be repeated twice* or some similar wording. This matter was referred to my head office at Pretoria and the following was received:

' "Sub-section 5 (*d*) of Section 65 of the Medical Dental and Pharmacy Act, No. 13 of 1928, provides that no more than one issue of the drug mentioned in any prescription for a habit-forming drug or order shall be made thereon. It is clear, therefore, that whether or not the prescriber (medical practitioner) adds the words *to be repeated twice* the person who dispenses such prescription may not issue the drug mantioned in such prescription more than once." '

'I will esteem it a great favour if this legal interpretation could be brought to the notice of the members of your Society.'

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

Cogentin Benztropine Methanesulfonate

Messrs. Mercke Sharpe & Dohme supply the following: Gogentin provides orally effective symptomatic and palliative treatment of all etiologic groups of Parkinson's syndrome—arteriosclerotic, idiopathic or postencephalitic.

It is indicated for the relief of parkinsonian tremor and rigidity and the amelioration of associated symptoms such as: sialorrhea, drooling, mask-like facies and pain due to cramps and muscle spasm. Cogentin is also of value when the patient is confined to bed and does not respond to other agents.

Dosage: Recommended daily dosage ranges from 0·5 mg. to 2 mg. administered orally, or 0·5 mg. initially followed by increments of 0·5 mg. until relief is manifest. Several days should lapse between increments to allow for cumulative effect. Younger patients with postencephalitic parkinsonism may be given 2 mg. two or three times daily. For older patients, 1 mg. twice daily is sufficient to control symptoms; a 2 mg. dose at bedtime will provide effective relief particularly when its effects are desired throughout the night. However, one dose each day is usually sufficient and more frequent doses may be unnecessary and undesirable.

Precautions: Since Cogentin is a cumulative drug of long action, continued supervision is desirable. Severe reactions such as mental confusion and excitement may occur with high dosage or in particularly susceptible individuals. Toxic effects, such as severe dryness of mouth, difficulty in swallowing, loss of weight and appetite or difficulty in speaking may also occur. Should such reactions become manifest, therapy with the drug should be temporarily discontinued and then resumed at a lower dosage.

Cogentin Methanesulfonate is supplied in bottles of 100 quarter-scored tablets, each tablet containing 2 mg. of the active ingredient, by Merck Sharp & Dohme International Division of Merck & Co., Inc., P.O. Box 5933, Johannesburg.

Three New B.D.H. Preparations.

This year at the 41st Medical Congress Exhibition of Pharmaceutical and Surgical Distributors, British Drug Houses (South Africa) (Pty.) Ltd., presented the '*Distaquaine' V (DQV)* range of products, the most recent and important advance in acid-stable oral penicillin therapy.

'Distaquaine' V is recommended in the treatment of all penicillin-sensitive infections where oral dosage is preferable. In order to obtain high levels of concentration in the blood of 5-20 units per ml., as is required in certain localized diseases such as osteomyelitis, and hitherto only reliably achieved by frequent parental injection, 'Distaquaine' V is now available in scored tablets containing 60 mg., 120 mg. and 240 mg. of phenoxymethylpenicillin.

The 'Distaquaine' V Elixir, in bottles of 30 × 30 mg. doses, provides an alternative to the tablets, and 'Distaquaine' V Sulpha Tablets, effective against a wide range of micro-organisms, are also available.

'*Ancoloxin*' tablets containing meclozine dihydrochloride 25 mg. ('Ancolan') and pyridoxine hydrochloride 50 mg., in each tablet were also featured. 'Ancoloxin' is a new approach to the nausea and vomiting of pregnancy, and it has been found that the combination provides a logical, rapid and strikingly effective control of the nausea, with each drug acting by different mechanisms: symptomatically and physiologically.

Thirdly '*Pectamol*', a new type of cough suppressant discovered in the B.D.H. Research Laboratories, occupied a prominent place. Its active constituent Oxeladin, 10 mg. to the teaspoonful, has a selective action on the medullary centre controlling the cough-reflex. 'Pectamol' presents a palatable, non-toxic preparation free from constipating or other side-effects. The active constituent is non-habit-forming and not related to opium alkaloids or their derivatives, 'Pectamol' is therefore ideally suited to both children and adults.

Proponesin-Analgesic.

British Drug Houses announce the introduction of a new synthetic analgesic discovered in the B.D.H. Research Laboratories under the trade name Proponesin. The new substance is tolpronine hydrochloride and it is issued in tablets containing 100 mg.

As a result of clinical investigation and comparison with other analgesics in 38 centres throughout the United Kingdom, the following three main advantages of Proponesin were evaluated:

1. It was found to have a remarkably rapid action.
2. No contra-indications were observed and no constipating or other side-effects were experienced.
3. It proved invaluable for patients intolerant to aspirin preparations or showing no response to other analgesics.

Proponesin Tablets are of outstanding value in relieving pain due to headache, dysmenorrhoea, sinusitis, toothache and herpes zoster. It has also postponed the need for more powerful analgesics such as morphine or pethidine, in the early stages of cases with progressively severe pain.

The suggested dosage scheme is one or two tablets prescribed three or four times daily; each tablet should be swallowed whole and not held in the mouth or crushed.

Proponesin is available in bottles of 10, 50 and 250 tablets each containing 100 mg. of tolpronine hydrochloride.

PASSING EVENTS : IN DIE VERBYGAAN

Department of Surgery, Red Cross War Memorial Hospital, Rondebosch, Cape. A post-graduate lecture will take place at the above hospital in the hospital lecture theatre on Wednesday, 9 October 1957 at 5 p.m. Subject: The METABOLIC RESPONSE to NEONATAL SURGERY. Speakers: Dr. R. de Villiers, Prof. J. H. Louw.

Research Forum, University of Cape Town. The next meeting of the Research Forum of the University of Cape Town will be held on Wednesday, 9 October (*not* Wednesday, 2 October) in the A. Floor lecture theatre, Groote Schuur Hospital, Cape Town, at 12 noon. The speaker will be Mr. D. J. du Plessis, F.R.C.S. His subject will be 'The Problem of Mikulicz's Disease'.

New Journal. Under the auspices of the Canadian Medical Association a new quarterly, the *Canadian Journal of Surgery* is starting publication. The Association will co-operate in this and the first issue of the new journal will appear on 1 October 1957. The Editorial Board consists of the professors of surgery from the twelve Canadian medical schools, with Dr. R. M. James of Toronto, President of the Royal College of Physicians and Surgeons of Canada, as chairman of the Board. Publication will be under the general supervision of the Editor of the *Canadian Medical Association Journal.* Subscription has been set at $10.00 for the first year. Enquiries about the journal should be addressed to 'Canadian Journal of Surgery', C.M.A. House, 150 St. George Street, Toronto 5, Ontario.

Unie van Suid-Afrika. Departement van Gesondheid. Aangifte van ernstige epidemiese siektes en poliomiëlitis in die Unie gedurende die tydperk 13 September tot 19 September 1957.

	Poliomiëlitis				
	Bl.	*Nat.*	*Kl.*	*As.*	*Totaal*
Transvaal	—	—	—	—	—
Kaapprovinsie ..	—	—	—	—	—
Oranje-Vrystaat ..	—	—	—	—	—
Natal	1	—	—	—	1
	—	—	—	—	—
Totaal ..	1	—	—	—	1

Pes, Pokkies, Tifuskoors: Geen.

REVIEWS OF BOOKS : BOEKRESENSIES

BLACK MAGIC AND WHITE MEDICINE

Black Magic and White Medicine. A Mine Medical Officer's Experiences in South Africa, the Belgian Congo, Sierra Leone, and the Gold Coast. By Michael Vane. Pp. 254. 5 Illustrations. 16s. London and Edinburgh: W. & R. Chambers Ltd. 1957.

Contents: South Africa. Belgian Congo. Sierra Leone. Gold Coast.

The author has had an immense amount of experience in examining and treating African labourers in South Africa, the Congo, Sierra Leone and the Gold Coast. In all these places he shows himself to be a humane and observant doctor who overcomes his difficulties with good humour and imperturbability. He tells of amusing encounters with malingerers, of witch doctors, and of the strange and inexplicable effects they produce in African patients even by 'remote control'.

His comments on the unreliability of Native nurses and nursing orderlies, culled as they are from 4 widely separated parts of Africa, seem to stress the similarity of the Natives' way of thinking and are not by any means unique. He points out how well the routine work is carried out if properly supervised but how disorganization takes place should an emergency arise.

The book emphasizes the long way the black man still has to go before he can hope to undertake the responsible tasks of the white man with success.

The difficulties of administration and the trials of having to persuade a primitive people that certain forms of therapy are necessary will be familiar to most South African doctors.

This book provides further evidence (if this be needed) of the lack of integration of the Bantu mind with modern thought.

S.T.

ORTHOPAEDIC MEDICINE

Text-Book of Orthopaedic Medicine. Volume I. Diagnosis of Soft Tissue Lesions. By James Cyriax, M.D. (Cantab.), M.R.C.P. (Lond.). Pp. xiv + 711. 36 Plates. 131 Figures. 45s. London: Cassell and Company Ltd. 1957.

Contents: Preface. List of Plates. I. Medical Fallacies. II. Traumatic Inflammation. III. Referred Pain. IV. Neuritis and Pressure on Nerves. V. Non-specific Arthritis. VI. Diagnosis of Soft Tissue Lesions. VII. Head, Neck and Scapular Area. VIII. Thoracic Outlet. Jaw. Sterno-clavicular Joint. IX. Shoulder. Part I. X. Shoulder. Part II. XI. Shoulder. Part III. XII Elbow. XIII. Wrist and Hand. XIV. Thorax and Abdomen. XV. Lumbar Region. Part I. XVI. Lumbar Region. Part II. XVII. Lumbar Region. Part III. XVIII. Lumbar Region. Part IV. XIX. Sacro-iliac Joint, Buttock and Hip. XX. Knee. XXI. Leg and Ankle. XXII. Foot. XXIII. Anaesthesia and Analgesia. XXIV. Psychogenic Pain. XXV. Physician and Physiotherapist. Index.

Dr. Cyriax is one of the doyens of physical medicine, and his book, in its 3rd edition, reflects all the attributes of a pioneer; across every page blazes the zeal and fervour of the missionary seeking to convert the 'medical heathens'. He is also an iconoclast, and it afforded the reviewer pleasure to read the denunciation of 'fibrositis' and 'rheumatism' as the diagnoses of the intellectually destitute. Consequently, it is not surprising that the decided views the author has about most soft-tissue lesions are stated vigorously and dogmatically. These are couched in terms new and often difficult to understand. What is meant, for example, by the 'vibrant twang of muscle spasm' (p. 106)? He states that the head and face are formed from the upper two cervical segments; this is embryologically meaningless.

There is universal agreement that hydrocortone is a valuable ancillary weapon in the armamentarium of the orthopaedic surgeon and the specialist in physical medicine. The reviewer, unfortunately, has not had the same striking successes as claimed by Dr. Cyriax. Hydrocortone, however accurately given, cannot possibly cure the surrounding thickening, occasionably almost cartilaginous, of teno-vaginitis of any standing. The author states that the diagnosis between monarticular infective arthritis and tuberculous infection is sometimes difficult and yet he advocates the injection of hydrocortone intra-articularly where myocrisin fails. Where the diagnosis is in doubt, the use of intra-articular hydrocortone is a dangerous practice which can produce and has produced catastrophes.

One of the many surprising statements made is that the subscapularis muscle must be weakened and the infrapinatus muscle strengthened to prevent recurrent dislocation of the shoulder. The basic defect is known to be a tear in the anterior glenoid labrum, which can only be repaired by operation. The Bankart procedure or Putti-Platt capsulorrhaphy is successful in over 80% of cases.

The chapter on the lumbar region contain many new and unusual concepts. The inclusion of a bibliography would have enhanced the value of the book for reference purposes.

This book, the most comprehensive of its kind so far, will be used extensively by those who intend to specialize in physical medicine.

M.S.

MEDICAL SIGNS AND SYMPTOMS

Symptoms and Signs in Clinical Medicine. Sixth Edition. By E. Noble Chamberlain, M.D., M.Sc., F.R.C.P. Pp. vii + 508. 374 Illustrations, 19 in colour. 35s. post 1s. 6d. Bristol: John Wright & Sons Ltd. Medical Publishers. 1957.

Contents: I. The Routine of Interrogation and Examination. II. External Characteristics of Disease. III. The Respiratory System. IV. The Cardiovascular System. V. The Urinary System. VI. The Digestive System. VII. The Haemopoietic System. VIII. The Nervous System. IX. The Nervous System (continued). X. Fever. XI. The Examination of Sick Children. XII. Medical Operations and Investigations. XIII. Radiology. XIV. Clinical Pathology and Biochemistry. Glossary. Index.

Dr. E. Noble Chamberlain's introduction to medical diagnosis requires no introduction to medical students, for whom it is primarily intended. The popularity of this book is indicated by the fact that since its first appearance in 1936, 6 editions and 7 reprints have been issued.

The present edition follows the familiar pattern, with some revision to bring up to date particularly the sections on the cardiovascular and nervous systems. The first part of the book is concerned with history-taking and physical examination, and with

the aid of numerous photographs, most of which are of good quality, this field is very adequately covered. The emphasis is on physical signs and in this regard the photographs serve a useful purpose in assisting the memory and judgment of the student. Individual systems are discussed under the headings of symptoms, signs, and diagnosis. A certain amount of repetition is thus inevitable but this is desirable in a book intended to enable a fourth-year student to co-ordinate clinical teaching with text-book information. The chapter by Professor Norman B. Capon on the examination of sick children constitutes a useful attempt to bridge the gulf which exists in the mind of the student between adult and paediatric medicine.

This book can be highly recommended to all medical students as an assimilable and attractive introduction to their training in the medical wards.

A.S.

SPORTS INJURIES

Sports Injuries—Their Prevention and Treatment. By Donald F. Featherstone, with a Foreword by Sir Arthur Porritt, K.C.M.G., K.C.V.O., C.B.E. Pp. 204. 48 Illustrations (26 as plates). 35s. post 1s. Bristol: John Wright & Sons Ltd. 1957.

Contents: Preface. Foreword by Sir Arthur Porritt, K.C.M.G., K.C.V.O., C.B.E. I. The Incidence and Type of Injury. II. The Prevention of Injuries. III. Examination and Diagnosis. IV. Methods of Treatment. V. Techniques of Treatment. VI. Injuries to the Knee-joint. VII. Foot and Ankle Injuries. VIII. Muscular Strains. IX. The Treatment of Contusions. X. Fractures and Dislocations. XI. The Miscellaneous Injuries of Sport. XII. Conclusions. Appendix: The Formation of a Treatment Room. Index.

This book by Donald Featherstone deals with sport injuries in a concise and workmanlike manner. It is a strange phenomenon that in this country of ours, where sport plays such a prominent part, we find so few books which deal specifically with this subject.

A point which strikes one immediately on reading this well-printed book is the fact that the author never tries to belittle the medical man. To him the doctor or orthopaedic surgeon is the master who controls the diagnosis and treatment, and the physiotherapist carries out this treatment and gives excellent and practical advice.

Mr. Featherstone is obviously an ardent follower of Dr. Cyriax' methods of treatment (like most medical men and orthopaedists who deal extensively with sport injuries). He favours the injection of local anaesthetic and early active and passive movements, and his suggestions of treatment have been found by the reviewer to be most effective.

Another point ably made is the value of quadriceps exercises in knee injuries and, above all, the need for getting the player on to his feet as quickly as possible in all injuries and for encouraging active exercises and, so far as possible, participation in games with his fellow players.

Psychology plays a big part in his treatment of injuries. His case histories are well set out and show the rapid recovery of the professional player who depends on his fitness for his bread and butter, whilst at times the amateur is inclined to exaggerate the seriousness of the injury. He also stresses the value of the doctor's telling the player the probable diagnosis immediately, and instituting treatment as soon as possible.

The foreword is by Sir Arthur Porrit, F.R.C.S., himself a fine sportsman and one of those who, in the past 25 years, have put St. Mary's Hospital on the map.

L.B.

HALE-WHITE

Hale-White's Materia Medica Pharmacology and Therapeutics. Thirtieth Edition. By A. H. Douthwaite, M.D. F.R.C.P. (Lond.). Pp. viii + 499. 24s. net. London: J. & A. Churchill Ltd. 1957.

Contents: Definitions. Pharmacy. Pharmaceutical Processes. Weights, Measures, Symbols. Pharmacopoeial Preparations. Administration of Drugs. Prescribing. Dangerous Drugs Act 1951, and Poisons Rules (1935). Pharmacological and Therapeutic Actions. 1. Demulcents, Emollients, Oleates and Dusting Powders. 2. Bitters. 3. Votatile Oils. 4. Digestive Ferments. 5. Purgatives. 6. Vegetable Astringents. 7. Cholagogues. 8. Anthelmintics. 9. Antiseptics. 10. Colouring Agents. 11. Substances whose Action is Physical. 12. Dyes. 13. Charcoal. 14. Water and Salt. 15. Sugars and Flavouring Agents. 16 Alkalies, Hydrates and Carbonates, Acetates and Citrates, Ammonium. 17. Saline Diuretics. 18. Calcium and Barium. 19. Acids. 20. Urea and Suramin. 21. Carbon Dioxide, Oxygen and Helium. 22. Iodides and Other Iodine Compounds. Drugs Acting on the Nervous System. 23. Bromides. 24. Alcohols. 25. Volatile Anaesthetics. 26. Hypnotics. Barbiturates and Other Drugs used in the Treatment of Epilepsy. 27. Opiates. 28. General Cerebral Stimulants. 29. Parasympatholytic Drugs. 30. Drugs acting chiefly on Autonomic Ganglia and Voluntary Muscle. 31. Local Anaesthetics. 32. Parasympathomimetic Drugs. 33. Sympathomimetic Drugs. 34. Sympatholytic Drugs. 35 Nitrates. 36. Antipyretics. 37. Cinchona, Pamaquine, Mepacrine. Drugs used in the Treatment of Malaria. 38. Salicylates and Benzoates. 39. Chemotherapeutic Agents. 40. Drugs used in Leprosy. 41. Antibiotics. 42. Digitalis, Strophanthus, Ouabain. 43. Emetics and Expectorants. 44. Drugs used in Amoebiasis. 45. Hydrocyanic Acid and Thiocyanates. 46. Colchicum, Phenylbutazone, Cinchophen, Probenecid. 47. Histamine and Antihistamines. 48. The Heavy Metals. 49. The Metalloids. 50. Hormone Preparations. 51. Antitoxins and Toxins. 52. Vaccines. 53. Vitamins. 54. Anticoagulants. 55. Insecticides and Insect Repellants. 56. Nitrogen Mustards. Appendix, Latin Phrases used in Prescriptions. Index.

This text-book has served many generations of students and practitioners since 1892; the original editor (Hale-White) was responsible for the first 19 editions, and the subsequent editor (Douthwaite) for 11 editions. In a book of this kind, special care is required in revision and with the accelerated changes that have taken place in pharmacology there is a great danger of incorrect facts being overlooked. In the present edition there is, as before, a great deal of information, particularly with regard to B.P. and B.P.C. drugs, systematically presented under individual titles, which will be of great value to those seeking data on official and approved drugs.

Among criticisms that must be made are the following: The classification and arrangement of drugs into satisfactory groups is not easy, but the close association of urea and suramin, of acetazoleamide and chemotherapeutic agents, of pethidine, methadone, and cannabis under page-titles of opiates is not good, nor is the subdivision of hormone preparations into class A and class B very helpful. The sub-title of action of drugs on nerve 'endings' is misleading. Two important typographical errors are stimulating for simulating (p. 123) and pyrosidine for pyridoxine (p. 452). The recommendation of strychnine for post-influenzal low blood pressure and as an aphrodisiac belongs to a past era. It should be emphasized that the symbols in the prescription on p. 17 are also obsolete.

The book has been maintained in its handy size and price.

N.S.

MATERIA MEDICA

The Essentials of Materia Medica Pharmacology and Therapeutics. Seventh Edition. By R. H. Micks, M.D., F.R.C.P.I. Pp. x + 432. 28s. net. London: J. & A. Churchill Ltd. 1957.

Contents: I. General Principles. II. Narcotic Action. III. Opium and Morphine. Other Potent Analgesics. IV. Non-Volatile Narcotics and Hypnotics. V. Anticonvulsants. VI. General Anaesthesia and Anaesthetics. VII. Convulsants and Analeptics. VIII. Adrenaline and Sympathomimetic Drugs. IX. Acetylcholine. Cholinergic Drugs. Neostigmine and Physostigmine. Organic Phosphorus Compounds. X. Acetylcholine Antagonists. Antispasmodics. Muscle Relaxants. Ganglion Blocking Drugs. The Treatment of Hypertension. The Chemistry of Muscle Relaxants and Ganglion Blocking Drugs. XI. The Antihistaminics. XII. Local Anaesthetics. XIII. Salicylates and Other Mild Analgesics. XIV. Drugs used in the Treatment of Heart Failure. XV. Diuretics. The Treatment of Oedema. XVI. Purgatives. The Treatment of Peptic Ulcer. XVII. Thyroid. Iodine. Antithyroid Drugs. Radio-active Iodine. XVIII. Calcium. Phosphorus. Parathyroid. Vitamin D. XIX. The Treatment of Anaemia. XX. Blood Clotting and Anticoagulant Therapy. XXI. Vitamins A, E and the B Complex. XXII. Gonadotrophic and Sex Hormones. Posterior Pituitary. Ergot. XXIII. Water and Electrolyte Depletion. XXIV. Insulin and Diabetes. XXV. Corticotrophin and Adrenal Cortical Steroids. XXVI. The Antibiotics. XXVII. The Sulphonamides. XXVIII. The Use of the Antimicrobial Drugs. XXIX. Metalloids and Heavy Metals. XXX. Antimalarial Drugs and Amoebicides. XXXI. Anthelmintics. XXXII. Cytotoxic Drugs. XXXIII. Prescribing. Index.

This book has proved popular in many quarters, because it deals in pleasant style with a limited number of important drugs and their application to therapeutics. The inclusion of a drug has depended on its value in the treatment of disease. It follows that many drugs are not considered and general information on drugs of traditional and wider use will need to be sought elsewhere. The text has been rewritten so as to give correct emphasis and perspective on therapeutic advances. A number of new drugs not described in previous editions are now presented, including such recent arrivals as oral hypoglycaemic drugs and 'academic' agents such as lysergic acid diethylamide. A strange name is that of 'hinconstarch', a combination of isoniazid and thiosemicarbazone built up on a carbohydrate polymer for the treatment of tuberculosis. For those responsible for intravenous infusions revised and (as is so often implied) lengthier information is provided on disturbances of water and electrolytes. There is a new chapter on anticonvulsant drugs.

With a pleasant format and easy style the book is even more than before a valuable clinical guide for the choice and use of drugs, with emphasis on the principles of pharmacology.

N.S.

PEPTIC ULCER

Gastro-Duodenal Ulcer. Physio-Pathology, Pathogenesis and Treatment. By J.-Jacques Spira. Pp. xvi + 549. 27 Figures. 82s. 9d. + 1s. 9d. delivery. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1956.

Contents: Preface. Introduction. *Part I. Physiological Considerations.* 1. Motor Activities of the Stomach and Duodenum. 2. Secretory Activities of the Stomach and Duodenum. 3. Co-ordination of Motor and Secretory Functions of the Stomach. 4. The Law of Isoperistalsis and its Corollaries. *Part II. Pathological Considerations.* 5. Pathological Anatomy of Peptic Ulcer. 6. Haemorrhage and Perforation. 7. Pain in Relation to the Peptic Ulcer Problem. 8. The Experimental Ulcer and its Significance in Relation to the Aetiology. *Part III. The Pre-Ulcerative Stage.* 9. The Significance of the Variations of the Gastric Secretions. 10. The Inflammatory Reaction of the Gastric Mucous Membrane. *Part IV. The Pathogenesis of Chronic Peptic Ulcer.* 11. The Theories of Chronic Ulcer Formation. 12. The Acid Factor in the Causation of Chronic Peptic Ulcer. 13. Factors Preventing Healing. 14. The Syndrome of Hyperfunction. 15. The Clinical Evolution of the Chronic Ulcer. 16. The Bile Factor in the Causation of Chronic Ulcer. 17. Author's Theory of Chronic Ulcer Formation. 18. The Role of Fat in the Economy of the Organism. 19. The Vitamins in Relation to Fat. 20. The Deleterious Effects of Fat. *Part V. Clinical Considerations.* 21. The Incidence of Peptic Ulcer. 22. Diagnosis. 23. Critique of Orthodox Treatment. 24. Treatment of Perforation and Haemorrhage. 25. The Author's Standard Treatment. Appendix. X-rays. References. Index.

It is Dr. Spira's belief that chronic peptic ulcer is caused by the combination of biliary regurgitation into the stomach and gastric hypersecretion. The former, he states, is due to an excess of fat in the diet, and his treatment of peptic ulcer is based on limitation of that substance. For this he claims excellent results, but his diet is bland and his standard programme includes the use of alkalies and sedatives, decreased physical activity, total avoidance of alcohol, and moderation in smoking.

The vast bulk of the book is a review of the literature of peptic ulcer; very little is devoted to the author's own opinions. The list of references takes up 170 pages. The text does not read easily because so much of it consists of quotations or paraphrases of statements that Spira feels tend to support his conclusions. One is struck by the lack of original work by the author, who nowhere lists the results of treatment of his own cases.

He lays greater stress on 'gastritis' as a pre-ulcer lesion than most modern workers would consider justified. Nor would all agree that acute and chronic ulcers are completely different in their development, or that gastric and duodenal ulcers are expressions of the same disease differing only in the site of the lesion. Inadequate attention is paid to the role of hormonal influences on gastric secretion and ulcer formation. The author does not belong to the psychosomatic school when it comes to the aetiology of ulcer.

One finds it difficult to believe, on the evidence presented, that Dr. Spira has in fact discovered *the* cause and *the* cure of chronic peptic ulcer.

D.M.K.

SURGICAL ANATOMY

A Synopsis of Surgical Anatomy. Eighth Edition. By A. Lee McGregor, M.Ch., F.R.C.S. With a Foreword by Sir Harold J. Stiles, K.B.E., F.R.C.S. (Edin.). Pp. xii + 808. 766 Illustrations by Dr. E. A. Thomas. 32s. 6d. post 1s. 2d. Bristol: John Wright & Sons Ltd. 1957.

Contents: Preface to the Eighth Edition. Preface to the First Edition. Foreword. Introduction. *Part I. Anatomy of the Normal.* I. The Scalp. II. The Meninges and Cerebrospinal Fluid. III. The Anatomy of the Normal and the Enlarged Pituitary Body. IV. The Thyroid and the Parathyroid Glands. V. The Tonsil. VI. The Breast. VII. The Umbilicus. VIII. The Gut. IX. The Peritoneal Fossae. X. Accessory Peritoneal Bands. XI. The Biliary Passages. XII. The Kidneys and Adrenals. XIII. The Triangle of Marcille. XIV. The Inguinal Region. XV. The Prostate. XVI. The Ischiorectal Fossa. XVII. The Anal Region. XVIII. The Vertebral Column. XIX. The Anatomy of the Child. XX. Nerves. XXI. Muscles. XXII. Fasciae. XXIII. Bones. XXIV. Joints. Tendon-Sheaths, and Bursae. XXV. Ligaments. XXVI. Veins. XXVII. Lymphatics. XXVIII Fat. XXIX. Lengths. XXX. Important Anatomical Levels. XXXI. Important Relations. *Part II. Anatomy of the Abnormal.* XXXII. The Anatomy of Congenital Errors. XXXIII. The Anatomy of Nerve Injuries. XXXIV. Bodily Habitus. XXXV. Anatomical Angles. Stiff Joints. XXXVI. Sphincters. XXXVII. Collateral Circulations. XXXVIII. The Teeth. XXXIX. The Limps of Infantile Paralysis. 'Snapping' Joints. XL. The Pathology of Bone in Terms of Anatomy. XLI. Rectal and Vaginal Examination. XLII. Anatomical Bases of Clinical Tests. XLIII. The Anatomy governing the Surgery of the Lymphatics. LXIV. The Anatomy governing the Surgery of the Symphathetic. XLV. The Anatomy of Certain Diseases. XLVI. The Anatomy of Surgical Procedures. XLVII. The Anatomy of Surgical Approach. Index.

This book was first published in 1932 and now the 8th edition has appeared. For an anatomy book this is indeed a remarkable record, but not one will find this surprising, because there are few surgeons who have not used this book with great benefit during their preparations for postgraduate degrees and afterwards.

The book is too well known to warrant a detailed description of its contents. Its object is to present anatomical facts of practical value without attempting to deal exhaustively with the anatomy of the whole body, and in this it succeeds admirably.

In this edition the book has been revised to bring it into line with present concepts; this is particularly noticeable in the sections on diaphragmatic hernia and the surgery of the sympathetic. Many additions have also been made, such as sections on paragangliomas, adrenalectomy and injuries to the large lymph ducts —these are written in the same lucid manner as the rest of the book.

There is no doubt that this book will (and should) continue to enjoy its tremendous popularity and it can be recommended confidently to all senior students and postgraduates in surgery. But more than that, every practitioner will find in this book valuable and interesting information and will be well rewarded by using it for occasional reading, for which it is so well suited.

D.J.duP.

LIVER DISEASE

Practical Diagnosis and Treatment of Liver Disease. By Carroll Moton Leevy, M.D. Pp. xii + 336. 84 Illustrations, including 23 in full colour. $8.50. New York: Paul B. Hoeber, Inc. 1957.

Contents: Foreword. Preface. 1. A Composite Approach. 2. Clinical Diagnosis. 3. Biochemical Liver Function Studies and Special Laboratory Procedures. 4. Anatomic Diagnosis. 5. Differential Diagnosis. 6. General Principles of Therapy. 7. Liver Disease Due to Nutritional Deficiency and Metabolic Disturbances. 8. Toxipathic Liver Disease. 9. The Liver in Circulatory Disturbances. 10. The Liver in Biliary Obstruction and Infection. 11. Neoplastic Disease of the Liver and Liver Trauma. Index.

This attractive volume reflects a heroic effort. The author has fitted his own store of clinical and laboratory data (derived over a period of years from a thousand patients with liver disease) into a background of the recent literature on the subject; out of this are evolved a manifold concept of liver pathology, a diagnostic compendium correlating bedside, biochemical and histological items, and a busy clinician's guide to the practical management of every type of listed disorder.

Like Berlioz in another sphere, the work falls short of greatness. In part this is due to an unexciting, compact style in a dominantly statistical key; but the writing is lucid and direct, and its succinctness is attained without solecism.

Errors of spelling or printing, trivial perhaps individually, occur in such profusion that irritation mounts in the pedantic reader, causing him at best to wonder how such a well-dressed book can be rushed through production so fast as to escape a final scrutiny, or at worst whether its very content can be entirely dependable. A list of such mistakes should interest the publishers rather than the prospective reader; but special comfort might be extended to several authors in the otherwise full and up-to-date reference-lists whose names receive a mauling, including Adlersberg on page 230, Budtz-Olsen (page 105), Dissè (page 302), A. B. Gutman (page 102), Himsworth (page 286), Niemann (page 225), and Thannhauser (page 231) who is said to have written 'Lipidosis' (ed. 2). In reference 26 on page 230 radio-iron is misspelt, and so is naphthalene on page 272. Syphilis suffers on page 270, and the discovery a few lines later of 'Chlonorchis senesis' vaguely suggests an elderly trematode rather than the Chinese liver-fluke which is meant.

Elsewhere in the text one flinches past 'hydrochloretics' and 'chloretics' (page 161) to describe drugs affecting bile-production, and three drugs in Table 16 (where toxipathic appears with an extra 'o') are also wrongly spelt. Towards the end the eruption of errors included pruritus masquerading as an inflammation (page 265), wrong vowels in residuum and vitamin (pages 268 and 279), periarteritis nodosa neutered in Fig. 45, caput medusa for caput medusae (page 282), and phosphorus looking like an adjective on page 238.

The index shares in this needless mutilation by having con-

sistently, in addition to several mis-spellings, the English amoebiasis where the text shows North American amebiasis, from which pattern it only departs to permit ameobiasis on page 324.

The substance of the book (lest the foregoing should savour of carping criticism) contains much that is useful. It is modern in its scope, slick yet searching in its academic chapters, and realistic in diagnostic and therapeutic programmes. Mention may be made of a pleasant section on hepato-renal relationships; of the emphasis placed on intolerance to surgical operations in those with impaired liver reserves; of the careful description of needle biopsy technique; and the uses and limitations of the procedure in many diseases involving the liver, including its value in the recognition of military tuberculosis. If the author is somewhat undiscerning in his account of anti-amoebic therapy, or of surgical procedures for oesophageal varices, he is encouraging in his personal experience of corticosteroids in the treatment of acute virus hepatitis, of certain instances of toxipathic liver disease, and of primary cholangiolar disease with threatening biliary cirrhosis not apparently due to infection. Students and practitioners, whose needs of the moment sometimes outweigh the luxury of enjoyment while reading, will find easy access to much organized material in this crisp monograph.

R.S.M.

REFRACTION

Practical Refraction. By B. C. Gettes. Pp. vi + 170 with 58 diagrams and illustrations. $6.50. New York: Grune & Stratton, Inc. 1957.

Contents: Preface. 1. Preliminaries to Sight Testing. 2. Astigmatism. 3. Astigmatism (Continued): Methods of Correction. 4. Accommodation. 5. Cycloplegia. 6. Subjective Methods of Testing. 7. Subjective Methods of Testing (Continued). 8. Hypermetropia. 9. Refraction of the Aphakic Patient. 10. Myopia. 11. Presbyopia. 12. Ophthalmic Lenses. 13. Contact Lenses. 14. Aids for Subnormal Vision. Index.

In concise, lucid terms, the author has in the compass of this short book succeeded in describing the art of refraction. Though intended for the instruction of the beginner with little or no previous experience, it nevertheless contains much of practical value and interest for the more advanced. Very correctly, the author has omitted the discussion of the geometry and physics of optics and the theory of retinoscopy that usually appear in a text-book on this subject, but has concentrated entirely on the practical side of refracting and the prescribing of spectacles.

He details the various procedures such as fogging, the use of the cross cylinder and so forth, and stresses the importance of the use of drugs when accuracy is essential, and for the pre-school and older child. Homatropine is a very unreliable and inconsistent cycloplegic and a good case has been made out for the use of cyclogyl. While agreeing that there is no reason why a myope should not have his full correction for constant wear, he recommends that bifocals are indicated regardless of age if that myope finds full, visual acuity essential for distance and full correction not tolerable for near.

For this country, his table of presbyopic aids appears too low. There is probably a climatic or racial reason for this, for it is well known that presbyopic symptoms develop later in life in inhabitants of the northern climates in comparison with those in the tropics. Apparently ophthalmic surgeons require presbyopic corrections early.

This book, then, while not as complete as, for instance, Duke-Elder's small book on 'The Practice of Refraction', serves a very useful purpose and makes a difficult subject appear extremely simple indeed.

L.S.

CANNED FOODS

Canned Foods—An Introduction to their Microbiology. Fourth Edition. By J. G. Baumgartner and A. C. Hersom, B.Sc., A.R.I.C. Pp. 291. 35 Illustrations. 21s. net. London: J. & A. Churchill Ltd. 1956.

Contents: I. Bacteria. II. True Fungi. III. Control of Spoilage. IV. Containers. V. Outline of Canning Operations. VI. Sources and Control of Contamination. VII. Principal Spoilage Organisms in Canned Foods. VIII. Effects of Heat on Micro-Organisms. IX. Principles of Canned Food Processing. X. Types of Spoilage. XI. Microbiology of Sound Canned Foods. XII. Bacterial Food-Poisoning. XIII. Laboratory Examination of Canned Foods. XIV. Examination of Raw Materials, Plant and Miscellaneous Methods. XV. Examination of Cans. Appendix. Index.

One is delighted to see this new edition of Baumgartner's 'Canned Foods'. It is the 4th edition of this excellent work, which was first published in 1943, under the authorship of J. G. Baumgartner, then senior bacteriologist of Messrs. Crosse and Blackwell Ltd., of London. In his new edition, Mr. Baumgartner, now factory control manager, is assisted as joint author by Mr. A. C. Herson, the present senior bacteriologist of the Company. Since its earlier edition, the work has undergone considerable revision, and the authors are to be congratulated on the concise manner in which they have dealt with the relevant material.

Industrial and medical microbiologists, factory control chemists, factory managers, medical officers of health, health officials and students of food technology, will find sufficient information in the pages of this book to provide them with an excellent background to canned foods, their processing, and the microbiological spoilage which they from time to time undergo.

For factory control personnel, the chapters dealing with control of micro-organisms, containers, the outline of canning operations, and the control of contamination, are recommended. For the microbiologist, the chapters dealing with the type of spoilage, the microbiology of sound canned food, bacterial food poisoning, and the laboratory examination of canned food, provide a valuable reference. The chapters dealing with the examination of cans as well as the summary of common seam defects will be of considerable value to all workers.

The authors have also dealt with the development of the aseptic method of canning, with the application of antibiotics, and with ionizing radiation in food processing, and they have reviewed the new techniques for heat processing.

In my opinion this book is a 'must' for all persons interested in the field of food canning.

W.R.M.

DISTURBANCES OF WRITING AND READING

Die Störungen der Schriftsprache (Agraphie und Alexie). Von Prof. Dr. A. Leischner. viii + 288 Seiten. 47 Abbildungen. DM 30.-. Stuttgart: Georg Thieme Verlag. 1957.

Inhaltsverzeichnis: Einleitung. Die Störungen des Schreibens. I. Die Entwicklung der Schrift. II. Die Einteilungen der Agraphien. III. Das klinische Syndrom der Agraphie. IV. Die Kasuistik der eigenen Beobachtungen zur Störung der Schriftsprache. 1. Teil Agraphie. V. Uberzicht über die eigenen Erfahrungen im klinischen Erscheinungsbilde der Agraphie. VI. Die Topik der Störbarkeit des Schreibens nach dem Schrifttum. VII. Die Untersuchung des Schreibens. Die Störungen des Lesens. VIII. Die Einteilung der Alexien. IX. Die Symptomatologie der Alexien. X. Die Kasuistik der eigenen Beobachtungen zur Störung der Schriftsprache. 2. Teil Alexie. XI. Ubersicht über die eigenen Erfahrungen im klinischen Erscheinungsbilde der Alexie. XII. Die Lokalisationsfrage der Alexie nach dem Schrifttum. XIII. Die Lokalisation der Schädigungen der Schriftsprache beim eigenen Krankengut. XIV. Die Untersuchung des Lesens. XV. Die Ätiologie der Störungen der Schriftsprache. XVI. Die Behandlung der Störungen der Schriftsprache. XVII. Das Wesen der Schriftsprache und ihrer Störungen im Spiegel des Schrifttums. XVIII. Eigene Stellungnahme zum Wesen der Agraphie und Alexie und zur Möglichkeit, diese Störungen zu lokalisieren.

This book treats of agraphia—loss of the faculty of writing—and alexia—failure to understand the written word.

The author has written a very comprehensive book about the subject, which has been, and still is, most controversial. He enumerates the classifications which various workers have put forward, and points out that the best classification is the one which takes into consideration that writing is a secondary cerebral function of fairly late development resulting from surroundings and education.

He sets out the clinical symptomatology of the agraphias, and then discusses 30 cases of his own and comes to the conclusion that a pure agraphia does not exist. The attempts by various workers to define a centre in the brain for this disturbance are, in his opinion, erroneous, because writing is phylogenetically a very young entity and by no means universal, consisting of a great number of different elementary components, with different cortical localization.

The part of the book devoted to the disturbances of reading again deals with classification, symptomatology, experiences with a number of the author's own cases, and a review of the probable localization. The author comes to the conclusion that there are primary and secondary brain functions. The primary

functions are contained in the original build of the brain and have their own executory organs. The secondary functions are accessory brain functions which only developed at a time when the development of the brain had already been completed. These therefore only have a loose connection with special areas—a relative localization which is dependant on education. Reading and writing are typical secondary brain functions.

The book is well written and not only gives the author's views on this debatable subject but also a very exhaustive survey of the literature. It is recommended for anyone who is particularly interested in the neurological aspect of this subject.

H.deV.H.

MAINGOT'S ABDOMINAL OPERATION

The Management of Abdominal Operations. Vols. I and II. Second Edition. Edited by Rodney Maingot, F.R.C.S. Pp. xvi + 1,432. Illustrations 540, with 444 figures. £8 net. London: H. K. Lewis & Co. Ltd. 1957.

Contents: Volume I. Preface. *Part I. General Considerations.* 1. Conditions affecting the Operative Risk for Abdominal Cases, by F. Avery Jones. 2. Preparation of the Good-Risk Patient, by Rodney Maingot. 3. Preoperative Treatment of the Acute Abdomen, by Rodney Smith. 4. Anaesthesia for Abdominal Operations, by J. Alfred Lee. 5. Remarks on Postoperative Care after the Uncomplicated Abdominal Operation, by Rodney Maingot. 6. Principles and Practice of Blood Transfusion, by R. J. Drummond. 7. Surgical Shock, by Rodney Smith. 8. Venous Thrombosis and Pulmonary Embolism, by Sol M. Cohen. 9. Fluid and Electrolyte Balance (Water Balance), in Surgical Patients, by L. P. le Quesne. 10. Postoperative Chest Complications, by Norman R. Barrett. 11. Complications of the Wound, by Ronald Reid. 12. The Use of Chemotherapeutic and Antibiotic Drugs in Abdominal Surgery, by William A. R. Thomson. 13. The Rôle of Vitamins in Abdominal Surgery, by Fred Wrigley. 14. Postoperative Parotitis, by Cecil Wakeley. 15. Genito-Urinary Tract Complications following Abdominal Operations, by J. C. Ainsworth-Davis. 16. Uraemia in Abdominal Surgery, by Clifford Wilson. 17. Alkalaemia and Acidaemia with Special Reference to Abdominal Cases, by Graham M. Bull. *Part II. Regional Considerations.* 18. Cancer of the Stomach, Cardia and Oesophagus, by John Borrie. 19. Lesions of the Oesophagus and Cardia, excluding Cancer, by John Borrie. 20. Gastric and Duodenal Ulcer, by Rodney Maingot. 21. The Complications and Sequelae of Gastric Operations, by Rodney Maingot. 22. The Treatment of Patients with Ulcer Haemorrhage, by Norman C. Tanner. 23. Acute Perforated Ulcer, by Norman C. Tanner. 24. Vagotomy, by H. Daintree Johnston. 25. Postgastrectomy Syndrome, by William W. Hallwright. *Volume II. Part II. Regional Considerations (Continued).* 26. Infantile Pyloric Stenosis, by David Levi. 27. Duodenal Ileus: Gastric Volvulus: Gastric and Duodenal Diverticula, by Rodney Maingot. 28. The Management of Biliary Tract Disease, by Rodney Maingot. 29. The Care of the Jaundiced Patient, by Ian Aird. 30. The Management of Cirrhosis of the Liver, by L. J. Witts. 31. Surgical Diseases of the Liver, by Cecil Wakeley. 32. The Surgical Aspects of Pancreatic Disease, by E. G. Muir. 33. Surgery of the Spleen, by R. Milnes Walker. 34. Inguinal Hernia, by A. K. Monro. 35. Strangulated Hernia, by A. K. Monro. 36. Intestinal Obstruction, by G. Qvist. 37. Chronic Appendicitis, by Rodney Maingot. 38. Acute Appendicitis, by A. J. Cokkinis. 39. Peritonitis: Primary and Secondary, by A. J. Cokkinis. 40. Post-operative Ileus, by A. J. Cokkinis. 41. Subphrenic Abscess, by H. R. S. Harley. 42. The Management of Surgical Lesions of the Colon, Rectum and Anus, by Henry R. Thomson. 43. The Management of Abdominal Injuries in Warfare and in Civil Life, by Gordon Gordon-Taylor. 44. The Management of Abdominal Operations in Children, by Denis Browne. *Part III. Special Considerations.* 45. Normal Values in Clinical Biochemistry, by D. N. Baron. Index of Names. Index of Subjects.

This, the 2nd edition of this well-known work, has been greatly improved by the substitution of two volumes for the original somewhat ungainly tome which made its appearance in 1953. Without exception, the authors are experts in their fields and have maintained a uniform standard of excellence under the editorship of Mr. Rodney Maingot. The surgeon of to-day has perforce to be no mean physiologist and the major portion of the first volume deals almost exclusively with the various physiological considerations relating to the proper care of the abdominal case both before and after operation. Fluid and electrolyte balance is of course dealt with in some detail, but it is surely not necessary to explain the significance of milli-equivalents in three different chapters. If the reader does not get the idea the first time, there is no reason to suppose that he will be any more successful on subsequent occasions. The chapters dealing with venous thrombosis and anaesthesia for abdominal cases are especially excellent, while the section concerned with chemotherapy and the antibiotics is noteworthy for its failure to deal with the danger of staphylococcal enteritis following the indiscriminate use of the antibiotics. The 2nd volume deals with regional considerations, and in this part the indications for various operative procedures are discussed, and an account given of the different operative techniques employed. Since this is a book dealing essentially with the care of the patient before and after operation, surgical technique is not described in any detail and the impression is gained that, in a work of this nature, it might with advantage have been dispensed with almost entirely. These are however minor criticisms. The book is a mine of information and well deserves a place on the bookshelf of the up-to-date surgeon and surgical trainee.

S.B.B.

RELIGIOUS FACTORS IN MENTAL ILLNESS

Religious Factors in Mental Illness. By Wayne E. Oates. Pp. xv + 239. 16s. net. London: George Allen & Unwin Ltd. 1957.

Contents: Preface. Acknowledgements. 1. The Hindering and Helping Power of Religion. 2. Self-Deception and Self-Encounter in the Religion of the Mentally Ill. 3. Religions Culture in the Making and Breaking of Personality. 4. Some Differences Between Healthy and Unhealthy Religion. 5. Interpersonal Relatedness and Religious Experience. 6. The Positive Work of Religion in the Therapy of Mental Illness. 7. The Psychiatrist's Approach to Religious Experience in Mentally Ill Patients. 8. Therapeutic Problems in Relation to the Religion of the Mentally Ill. Appendix: Standards for Clinical Pastoral Education Adopted by the National Conference on Clinical Pastoral Training October 1, 1952.

People who have to deal with mentally disordered patients frequently encounter problems in which it is difficult to say whether they are dealing with psychiatric or religious questions, and find it difficult at times to decide at what point a religious idea becomes abnormal or whether the rather peculiar religious ideas harboured by some individual can be regarded as acceptable to his church or denomination.

In this book Dr. Oates sets out to answer these problems. He differentiates between true religion and religious ideas used as 'a self-concealment device'. He points out, too, the fallacies and misinterpretation of ideas that are found in the religious life of many who must be regarded as normal.

Dr. Oates makes a strong plea for better co-operation between the medical profession and the clergy, and in doing so he discusses the attitude of various psychiatrists towards religion and that of certain ministers of religion towards psychiatry. He gives very fair and careful consideration to defining the respective fields of influence and so avoiding trespass on each others preserves, but even so he advocates pastoral psychotherapy to a much greater degree than we are accustomed to or than we should expect from a non-specializing minister. Nevertheless Dr. Oates puts up a very good case for more intelligent co-operation between the doctor and the parson, in which each recognizes the other's qualifications and respects his clinical ability and insight rather than exhibiting a polite and benevolent tolerance of each other.

Unfortunately the psychological technicalities so often found in American works of this type make the reading of this book a difficult and involved matter.

D.S.H.

CORRESPONDENCE : BRIEWERUBRIEK

SYSTEMATIC DESENSITIZATION IN PSYCHOTHERAPY

To the Editor: I read the article on psychotherapy by Drs. Lazarus and Rachman in your issue of 14 September with great interest. In view of the promise held out by the new techniques described by Wolpe in earlier articles, it is encouraging to obtain independent substantiation of the value of these methods. Apart from other considerations, they appear to provide a simplified and quicker means of therapy than that offered by psycho-analysis.

There is one aspect of this therapy which does seem to require elaboration however. The concept of 'reciprocal inhibition' as originally employed, refers to a relatively uncomplicated and specific neural activity. Is it possible to apply this essentially physiological concept to behaviour in this manner?

E. Schultz

Baragwanath Hospital
Johannesburg
19 September 1957.